

This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`



LANE



MEDICAL LIBRARY

LEVI COOPER LANE FUND


LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
300 PASTEUR ROAD
PALO ALTO, CALIFORNIA



INJURIES & DISEASES
OF THE
BONES AND JOINTS

INJURIES & DISEASES OF THE BONES AND JOINTS

THEIR DIFFERENTIAL DIAGNOSIS BY MEANS OF THE ROENTGEN RAYS

BY

FREDERICK H. BAETJER, M.D.

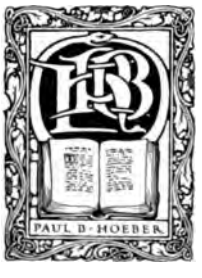
Associate Professor of Roentgenology, Johns Hopkins University; Roentgenologist, Johns Hopkins Hospital

AND

CHARLES A. WATERS, M.D.

Instructor in Roentgenology, Johns Hopkins University; Assistant Roentgenologist, Johns Hopkins Hospital

ILLUSTRATED WITH 332 ROENTGENOGRAMS AND ONE LINE DRAWING



LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305

NEW YORK
PAUL B. HOEBER
1921

COPYRIGHT, 1921
By PAUL B. HOEBER

ALL RIGHTS RESERVED

Published, March 1921

LANE LIBRARY

Printed in the United States of America

To

WILLIAM STEWART HALSTED

Dear Dr. Halsted,

Your kind consent to accept the dedication of this book has brought to a happy conclusion our labors of the past year.

Your sympathy and encouragement have been a constant stimulus in the study of the many problems which have confronted us.

We offer to you this, our first book, as a token of the esteem and gratitude in which we hold you as one who has ever represented, not only to us but to the profession at large, the best ideals in surgery.

Very sincerely yours,

F. H. BAETJER
C. A. WATERS

February 28, 1921.

51168

PREFACE

THE TECHNICAL side of roentgenology has been so ably described by various authors that mention of it is mere repetition, therefore, we have in this book, confined ourselves to the study of the finished product—the plate.

Our object then, is to show that roentgenology is not a picture process, but a medical procedure based upon careful analysis and logical deductions from the shadows observed upon a plate and the translation of these shadows into pathological terms.

The constituent parts of the normal bones and their functions have been discussed; and with these as a basis, the lesions have been studied and correlated with the gross pathological findings of the operating and autopsy rooms.

Age and sex play such an important rôle in the production, character and situation of the lesion that we have linked them up with the *x*-ray plate in arriving at a diagnostic conclusion.

Inasmuch as the pathological findings and classifications in certain types of bone and joint lesions are not yet thoroughly established, we may state that in the same way our interpretations of the *x*-ray shadows of these lesions also may be subject to revision in the light of further pathological investigations.

No attempt has been made to give a bibliography, as this book is the result of our observations in the clinic of the Johns Hopkins Hospital. In this connection we wish to express our appreciation for the assistance given us by the x-ray Staff of the Johns Hopkins Hospital.

In conclusion our thanks are also due to Mr. Paul B. Hoeber and his editorial staff for their painstaking care and efficient cooperation in the preparation of this volume.

F. H. B.
C. A. W.

February 28, 1921.

CONTENTS

CHAPTER		PAGE
I	INTRODUCTION	3
II.	NORMAL BONES. The Periosteum, The Cortex, The Medullary Canal, The Nutrient Foramen, Cartilage, Joints, Growth, Joint Lesions, Effect of Sex.	13
III.	EPIPHYSES. Scapula, Clavicle, Humerus, Radius, Olecranon, Carpal Bones, Phalanges, Pubis and Ischium, Femur, Patella, Tibia and Fibula, Vertebrae.	33
IV.	FRACTURES OF THE UPPER EXTREMITIES. Elements Affecting Fractures—Muscular Tension, Age and Sex, Occupation, Condition of Soft Tissues, Bone Atrophy, Formation of Callus. Fractures from Pathological Causes, Fractures Classified According to Age—The Skull, Facial Bones, Mandible, Clavicle, Scapula, Humerus, Forearm, Wrist, Hand, Ribs, Sternum, Pelvis.	47
V.	FRACTURES OF THE LOWER EXTREMITIES. Femur, Patella, Tibia and Fibula, Bones of the Foot, Astragalus, Os Calcis, Scaphoid, Cuboid and Cuneiforms, Metatarsal Bones, Phalanges, Sesamoids, Bone Splints.	99
VI.	CONGENITAL DISLOCATIONS. Congenital Dislocations of the Hip, Dislocations of the Shoulder Joint, Abnormalities in the Development of Epiphyses.	133
VII.	ACQUIRED DISLOCATIONS. Shoulder, Elbow, Ulna and Radius, Wrist, Hand, Pelvic Bones, Hip, Patella, Knee, Tibia, Fibula.	145
VIII.	BONE INFECTIONS. Osteomyelitis—Tuberculosis, Lues. Typhoid, Actinomycosis, Raynaud's Disease, Leprosy, Coccidoidal Granuloma, Mineral Poisoning, Special Infections.	159
IX.	JOINT LESIONS IN CHILDREN. Rickets, Congenital Lues, Scurvy, Tuberculosis, Acute Epiphysitis—Non-tuberculous, Perthes's Disease or Juvenile Deforming Osteochondritis.	183
X.	JOINT LESIONS IN ADULTS. Changes Indicating an Arthritic Condition, Acute Polyarticular Rheumatism, Chronic Arthritis—Tuberculosis, Caries Sicca, Luetic Arthritis, Atrophic, Hypertrophic Arthritis. Gonorrheal Arthritis, Arthritic Changes due to Age, Arthritis in the Spine, Villous Arthritis, Non-arthritic Joint Lesions—Gout, Charcot Joint, Syringomyelia. Hemophilia, Arthritic Changes in Ligaments.	205

CHAPTER PAGE

XI. BONE TUMORS 241
Method of Analyzing Bone Tumors, Carcinoma, Hypernephroma, Round-cell Sarcoma, Spindle-cell Sarcoma, Periosteal and Osteosarcoma, Myeloma, Giant-cell Sarcoma, Enchondroma or Osteochondroma, Cyst, Osteoma, Fibroma, Myxoma, Hemangiomata, Ossifying Hematoma, Osteitis Fibrosa Cystica, Brain Tumors.

XII. THE SPINE. 289
Conformation, Divisions, Classification of Lesions, Diagnostic Aids, Arthritis, Infectious Arthritis, Acute Spondylitis Deformans, Charcot Spine and Syringomyelia, Tumors, Application of Classification Aids, Functional Conditions.

XIII. ABNORMALITIES 311
Extra Ribs, Non-union—Rudimentary Vertebrae, Abnormalities in the Lumbosacral Region, Clavicle, Scapula, Shoulder, Forearm, Hand, Femur, Tibia and Fibula, Ankle and Foot.

XIV. DYSTROPHIES 323
Acromegaly, Osteomalacia, Chondrodystrophies, Pulmonary Osteoarthropathy, Osteogenesis Imperfecta, Dyschondroplasia.

LIST OF ILLUSTRATIONS

FIGURE		PAGE
1.	Section of normal radius and ulna with periosteum invisible	14
2.	Deposition of calcium salts in the periosteum of the tibia	15
3.	Bony cortex of the tibia	16
4.	Finely reticulated bony structure at the end of the bones	17
5.	Medullary canal in the long bones	18
6.	Joint cartilage	19
7.	Cartilage present in costal cartilage	19
8.	Tibia and fibula showing cortex, medullary canal, and cancellous bone	20
9.	Flat bone with grooves for blood vessels visible	20
10.	Carpal bone	20
11.	Normal joint space filled with cartilage	22
12.	Destruction of cartilage in joint space	22
13.	Abnormally wide joint space at the knee in early childhood	23
14.	Wide epiphyseal lines in the wrist of a child of seven	23
15.	Narrowing of epiphyseal line of the first metacarpal in a child of eleven	24
16.	Head of humerus of a new-born child	25
17.	Atrophy in shoulder joint of a man of fifty	26
18.	Position of the sacrum in the female pelvis	28
19.	Position of the sacrum in the male pelvis	29
20.	Ossification of epiphyseal centers in a child of eighteen months	34
21.	Ossification of epiphyseal centers in a child of three years	34
22.	Delayed ossification of carpal bones in a child of ten years	34
23.	Ossification of the epiphysis of the acromion in a child of fifteen years	34
24.	Ossification of the head of the humerus in a child of four years	35
25.	Epiphysis at the elbow joint in the fourteenth year	36
26.	Lower epiphysis of the radius	37
27.	Epiphysis of the olecranon at the fifteenth year	37
28.	Centers of ossification of the carpal bones at four years	38
29.	Centers of ossification of the carpal bones at seven years	38
30.	Centers of ossification of the carpal bones at eleven years	38
31.	Pubis and ischium at five years	39
32.	Epiphysis of the greater trochanter at nine years	40
33.	Epiphysis of the lower end of the femur in child under six months	41
34.	Epiphyseal center of the patella at four years	42
35.	Well-developed head of the tibia at eight years	42
36.	Epiphyseal centers of the ankle joint present at birth	43

FIGURE PAGE
37. Three centers of ossification in the vertebrae 43
38. Location of the epiphyses of the first and other metacarpals 44
39. Swelling from fracture of the lower third of the radius 50
40. Old Colles's fracture showing marked atrophy 50
41. Callus formation six weeks after fracture 52
42. Backward displacement of the lower end of the humerus 52
43. V-shaped skull fracture with depression in the frontal region 55
44. Linear skull fracture in the occipital region 56
45. Fracture of the malar bone with hemorrhage into the sinus 57
46. Fracture of the mandible followed by osteomyelitis 58
47. Fracture of the condyle of the mandible with displacement 58
48. Fracture of the coronoid process of the mandible 60
49. Oblique fracture of the mandible, not complete 60
50. Green-stick fracture of the outer third of the clavicle in a child 61
51. Fracture of the outer third of the clavicle with slight displacement 62
52. Fracture of the extreme tip of the clavicle 63
53. Fracture of the body of the scapula 63
54. Fracture at the base of the spine of the scapula 64
55. Fracture and displacement beneath the glenoid fossa 65
56. Linear fracture of the coracoid process 65
57. Fracture of the tip of the acromial process 66
58. Epiphyseal separation of the upper end of the humerus 66
59. Fracture of the surgical neck of the humerus 67
60. Fracture of the head of the humerus with outward displacement 68
61. Fracture of tuberosity and surgical neck of the humerus with atrophy 69
62. Fracture of the surgical neck of the humerus 70
63. Old fracture of the greater tuberosity of the humerus 71
64. Epiphyseal separation of the lower end of the humerus 72
65. Green-stick fracture of the humerus 72
66. Spiral fracture of the humerus 72
67. Attempted reduction of supracondylar fracture of lower end of the humerus 73
68. Fracture of the external condyle of the lower end of the humerus 74
69. Fracture of the olecranon process 75
70. Fracture of the olecranon process 75
71. Fracture of the coronoid process 76
72. Fragment of the head of the radius 76
73. Fracture of the head of the radius 77
74. Fracture of the neck of the radius 77
75. Fracture of the upper third of the ulna—lateral view 78
76. Fracture of the upper third of the ulna—anteroposterior view 78
77. Old fracture of the upper third of the ulna 79
78. Green-stick fracture of the radius and ulna 79
79. Fracture and displacement of the radius and ulna 80

FIGURE PAGE
80. Backward dislocation of the epiphysis of the radius..... 80
81. Epiphyseal separation of the lower end of the radius..... 81
82. Fracture of both bones of the forearm at site of Colles's fracture 82
83. Fracture of the forearm at site of Colles's fracture..... 82
84. Colles's fracture of the forearm with marked displacement..... 83
85. Colles's fracture with anterior displacement of the lower fragment 83
86. Old Colles's fracture with backward displacement..... 84
87. Impacted Colles's fracture without displacement..... 84
88. Old Colles's fracture with angulation. No displacement..... 85
89. Barton's fracture..... 86
90. Old fracture of the styloid of the radius..... 86
91. Fracture of the scaphoid of the wrist without displacement..... 88
92. Colles's fracture associated with the scaphoid. No displacement 88
93. Fracture of the first metacarpal bone with displacement..... 89
94. Oblique fracture of a metacarpal bone without displacement.... 89
95. Fracture of the first metacarpal bone with angulation..... 89
96. Longitudinal fracture of the terminal phalanx of the thumb.... 90
97. Crushing fracture of the terminal phalanx with osteomyelitis... 90
98. Base-ball Finger..... 90
99. Fracture of several ribs with slight displacement..... 91
99a. Fracture of the sternum showing lateral displacement..... 92
100. Fracture of the upper third of the femur, ilium and pubis..... 93
101. Fracture of ischium and pubis..... 94
102. Fracture of the ilium by a crushing injury..... 95
103. Fracture of the acetabulum with head of femur in pelvic canal.. 96
104. The epiphyseal separation of the head of the femur..... 100
105. Fracture of the neck of the femur just behind the head..... 101
106. Old fracture of the mid portion of the neck of the femur..... 102
107. Intertrochanteric fracture..... 103
108. Absorption of the neck of the femur in old ununited fracture.... 104
109. Healed fracture of the neck of the femur without callus..... 105
110. Intertrochanteric fracture with coxa vara..... 106
111. Position of a fracture of the upper third of the femur..... 107
112. Green-stick fracture of the femur..... 108
113. Transverse fracture of the femur..... 108
114. Epiphyseal separation of the lower end of the femur..... 110
115. Old epiphyseal separation of the lower end of the femur..... 111
116. Oblique fracture of the lower end of the femur..... 112
117. Lateral view of Fig. 116 with line of fracture visible..... 113
118. Fracture of the patella..... 114
119. Fracture of the patella..... 114
120. Fracture of the tibial tubercle..... 114
121. Fracture of the tuberosity of the tibia..... 115
122. Fracture of the upper end of the fibula..... 116
123. Fracture of the external tibial spine..... 117

FIGURE PAGE
124. Green-stick fracture of the tibia in the first age period..... 118
125. Oblique fracture of the tibia in the first age period..... 118
126. Spiral fracture of the tibia in the first age period..... 118
127. Comminuted fracture of tibia and fibula..... 118
128. Epiphyseal separation of the lower end of the tibia..... 119
129. Fracture of the lower end of the tibia..... 119
130. Typical Pott's fracture, anteroposterior view..... 120
131. Lateral view of a Pott's fracture..... 120
132. Clear triangle in the soft parts at the normal ankle joint..... 121
133. Triangle obscured by swelling from injury or disease..... 121
134. Fracture of the astragalus..... 122
135. Fracture of the end of the astragalus..... 122
136. Fracture of the os calcis due to a crushing injury..... 123
137. Fracture of the tip of the scaphoid..... 123
138. Old fracture of the cuboid, with callus formation..... 124
139. Fractures of metatarsals and phalanges..... 124
140. Fracture of the base of the fifth metatarsal..... 125
141. Old fracture of the tibia with bone graft..... 126
142. Bone transplant of the upper end of the humerus..... 128
143. Pressure atrophy around the ends of the bone graft..... 128
144. Old fracture of the humerus with bone graft..... 129
145. Metal plate acting as an irritant..... 129
146. Tibia after a bone graft has been removed..... 130
147. Straight sides of the pelvis before walking..... 134
148. Beginning concavity of the sides of the pelvis..... 135
149. Congenital dislocation of the hip with shallow acetabulum..... 135
150. Destruction of the head of the femur with dislocation..... 136
151. Bony ankylosis seen in non-tuberculous infection..... 137
152. Undeveloped femur in congenital dislocation..... 138
153. Congenital dislocation of both hips with shallow acetabula..... 138
154. Partial subluxation of the ankle joint from injury..... 139
155. Club foot..... 139
156. Club hands..... 139
157. Abnormality of the femur due to anterior poliomyelitis..... 140
158. Subcoracoid dislocation of the humerus..... 146
159. Subglenoid dislocation with unusual position of the humerus..... 147
160. Subglenoid dislocation with usual position of the humerus..... 147
161. Subglenoid dislocation of the humerus associated with fracture..... 148
162. Dislocation of both bones of the forearm backward..... 149
163. Another view of Fig. 162 showing lateral displacement..... 149
164. Dislocation of the elbow..... 149
165. Lateral view of Fig. 164..... 149
166. Dislocation of the elbow reduced..... 150
167. Dislocation of the wrist joint..... 150
168. Anterior dislocation with rotation of the semilunar..... 150

FIGURE PAGE
169. Backward dislocation of the thumb phalanx ... 151
170. Backward dislocation of the hip ... 152
171. Obturator dislocation of the hip ... 153
172. Lateral dislocation of the patella ... 154
173. Subluxation of the tibia ... 154
174. Posterior dislocation of the foot ... 155
175. Dislocation of the first metatarsal ... 156
176. Osteomyelitis with involvement of medullary canal and cortical bone ... 160
177. Osteitis with an extensive periostitis ... 160
178. Acute infection in the joint involving the femur ... 161
179. Compound fracture with osteomyelitis ... 162
180. Osteomyelitis with sequestrum ... 162
181. X-ray of an acute osteomyelitis one week after onset ... 164
182. Chronic osteomyelitis with new bone formation ... 165
183. New bone formation showing boundaries of infection ... 166
184. Exterior deposition of bone simulating expanded shaft ... 169
185. Chronic osteomyelitis with sequestrum ... 168
186. Brody's abscess in the head of the radius ... 168
187. Typical tuberculosis of the shaft of the radius ... 169
188. Tuberculosis of one side of the epiphysis of the tibia ... 171
189. Tuberculosis of the first metacarpal with periostitis ... 172
190. Tuberculous osteomyelitis in an infant with sequestrum ... 172
191. Luetic periostitis of the forearm ... 173
192. Luetic periostitis of the ulna (lace work type) ... 173
193. Luetic osteomyelitis without swelling of the soft tissue ... 174
194. Involucrum with the entire shaft becoming a sequestrum ... 174
195. Acute osteomyelitis with no involucrum ... 175
196. A typhoid periostitis with a cavity in the cortex ... 176
197. Raynaud's disease showing appearance of terminal phalanges ... 177
198. Leprosy ... 178
199. Leprosy ... 178
200. Coccidoidal granuloma ... 179
201. Coccidoidal granuloma ... 179
202. Coccidoidal granuloma ... 179
203. Saucer-shaped expansion of the epiphyses in rickets ... 184
204. Saucer-shaped expansion of the epiphyses in rickets ... 184
205. Saucer-shaped epiphyses of tibia and fibula with atrophy ... 185
206. Atypical rachitic changes ... 185
207. Atrophy in rickets as the cause of multiple fractures ... 186
208. Atalectatic strips of consolidation in the chest from rickets ... 187
209. Rachitic condition shown by faulty calcification in epiphyses ... 189
210. Congenital lues in tibia and fibula ... 189
211. Periostitis present in congenital lues ... 190
212. Trümmer zone of destruction in scurvy ... 191

FIGURE PAGE
213. Elevation of periosteum and organized hemorrhage in scurvy... 191
214. Beginning organization of hemorrhage under torn-up periosteum 192
215. Ossifying hematoma in scurvy .. 193
216. Cod-liver oil feeding in rickets differentiated from scurvy .. 196
217. Rickets after cod-liver oil feeding .. 196
218. Healed rickets after cod-liver oil feeding .. 197
219. Tuberculosis of the knee with marked bazing and atrophy 198
220. Non-tuberculous infection with bone production .. 199
221. Juvenile deforming osteochondritis *Perthes's disease* .. 200
222. Juvenile deforming osteochondritis *Perthes's disease* .. 201
223. Graphic illustration of the stages of bone infection 206
224. Acute polyarticular arthritis .. 208
225. Infectious arthritis in the first stage 208
226. Second stage of infectious arthritis 210
227. Third stage of infectious arthritis ... 212
228. Tuberculous arthritis of the wrist ... 214
229. Tuberculous arthritis of the knee, almost healed 215
230. Caries sicca of the shoulder joint with atrophy 216
231. Gonorrheal arthritis of patella, tibia and femur 217
232. Luetic arthritis with periosteal changes 218
233. Atrophic arthritis with atrophy 219
234. Hypertrophic arthritis with exostoses and joint mice 220
235. Joint mice in hypertrophic arthritis 221
236. Synovial thickening in villous arthritis 224
237. Punched-out areas characteristic of gout in joints 226
238. Early case of gout with punched-out areas missing 227
239. Charcot joint with complete destruction. No atrophy 228
240. Anteroposterior view of Fig. 239 229
241. Charcot joint of the ankle with marked periostitis 230
242. Hemorrhage cysts beneath the cartilage of the joint 231
243. Hemophiliac joint with surfaces destroyed by hemorrhage 232
244. Organization of hemorrhage in elbow joint 232
245. Bone formation in the tendo Achillis from arthritic condition... 234
246. Deposition of salts in the subdeltoid bursa 235
247. Deposition of salts in the subdeltoid bursa 235
248. Non-gonorrheal type of exostosis of the os calcis 236
249. Gonorrheal exostosis 236
250. Tumor arising within the medullary canal. (Giant-cell sarcoma.). 244
251. Tumor arising from the cortex. (Fibromyxoma.) 245
252. Bone production within a tumor. (Osteoma.) 246
253. Benign tumor with slight expansion of the bone. (Cyst.) 248
254. Benign tumor of the fibula. (Cyst.) 249
255. Complete destruction of the head. (Round-cell sarcoma.) 250
256. Expansion of cortex from medullary tumor. (Giant-cell sarcoma.) 251

FIGURE PAGE
257. Metastatic carcinoma showing invasion ... 252
258. Metastatic carcinoma with pathological fracture ... 256
259. Carcinomatous metastases of the skull ... 257
260. Carcinoma of the mandible ... 258
261. Metastasis from a prostatic carcinoma ... 259
262. Miliary prostatic metastases of the lumbar vertebrae ... 260
263. Round-cell sarcoma of the humerus ... 261
264. Spindle-cell sarcoma of the tibia ... 262
265. Advanced case of periosteal sarcoma ... 264
266. Periosteal sarcoma with bone striae perpendicular to the shaft ... 264
267. Periosteal sarcoma with bone striae in the soft tissues ... 265
268. Osteosarcoma with dense bone production ... 265
269. Giant-cell sarcoma with cortex intact ... 266
270. Giant-cell sarcoma with spontaneous healing ... 267
271. Lateral view of Fig. 270 ... 267
272. Multiple enchondromata of medullary and cortical origin ... 268
273. Osteochondroma of the head of the fibula ... 269
274. Large osteochondroma of the femur ... 270
275. Bone cyst of the humerus, which is multilocular ... 272
276. Pathological fracture of the humerus through a bone cyst ... 272
277. Osteoma showing typical cauliflower growth ... 273
278. Osteoma ... 273
279. Table-top type of exostosis ... 274
280. Pencil type of exostosis pointing away from the nearest epiphysis ... 274
281. Exostosis terminating in osteochondroma ... 275
282. Fibroma of the phalanx ... 275
283. Hemangioma with calcified bodies in the soft tissue ... 276
284. Hemangioma of the hand ... 277
285. Ossifying hematoma of the femur ... 278
286. Myositis ossificans ... 278
287. Osteosarcoma with bone destruction ... 279
288. Osteitis fibrosa cystica with lengthening of the tibia ... 281
289. Pressure atrophy of the cranial bones from internal pressure ... 282
290. Destruction of the sella turcica from hypophyseal tumor ... 283
291. Centers of ossification of the vertebrae ... 290
292. Fracture and subluxation of the second cervical vertebra ... 292
293. Fracture of the transverse process of the lumbar spine ... 293
294. Scoliosis showing primary and secondary curves ... 294
295. Lateral angulation of the spine ... 295
296. A fractured spine with new bone production ... 296
297. Arthritis with edges of the vertebrae sharpened ... 298
298. Exostoses along the vertebrae and lateral ligaments ... 298
299. Destruction and fusion of two vertebrae with angulation ... 299
300. Marie Strümpell type of arthritis deformans ... 300

FIGURE PAGE
301. Charcot joint of the fourth lumbar vertebra. 301
302. Carcinoma of the lumbar vertebra with joint spaces intact. 303
303. Pressure destruction of a lumbar vertebra from a tumor. 308
304. Rudimentary vertebra arising from the seventh cervical vertebra 312
305. Cervical rib arising from one side only. 313
306. Rudimentary spina bifida of the seventh cervical vertebra. 314
307. Rudimentary vertebra in the dorsal region. 314
308. Synostosis of the bodies of two ribs. 315
309. Congenital non-union of the laminae of the sacrum. 315
310. A pair of lumbar ribs. 316
311. Sacrolization of the fifth lumbar vertebra. 316
312. An attempt at sacrolization on one side. 317
313. Aberrant articulation between clavicle and coracoid. 317
314. Absence of the ulna due to a faulty epiphysis. 318
315. Extra digit arising from the little finger. 318
316. Fourth and fifth fingers with common metacarpal. 319
317. Extra digit on the toe. 319
318. Congenital absence of the fibula. 319
319. Osteitis deformans. 324
320. Osteitis deformans. 324
321. Changes in cranial bones in osteitis deformans. 325
322. Acromegalic skull. 326
323. Changes in the bones of the hand in acromegaly. 327
324. Osteomalacia. 328
325. Osteomalacia. 328
326. Achondroplasia. 329
327. Achondroplasia with deformity of the radius. 330
328. Pulmonary osteoarthropathy. 331
329. Osteogenesis imperfecta with multiple fractures. 332
330. Osteogenesis imperfecta with multiple fractures. 333
331. Osteogenesis imperfecta with multiple fractures. 333

CHAPTER I

INTRODUCTION

INJURIES AND DISEASES OF THE BONES AND JOINTS

CHAPTER I

INTRODUCTION

ROENTGENOLOGY is of comparatively recent origin; only within the last decade and a half has it made its greatest progress. Before that period our entire energies were devoted to the purely mechanical side of developing a technique so that examinations could be properly made. Today one hears less and less of technique and more and more of diagnostic principles.

Unfortunately the term "picture" has been introduced, not in the sense that an internist would use it when he speaks of a clinical picture, but as one would speak of a photograph. There is no such thing as an *x*-ray picture. A roentgenogram is a projcetion upon a photographic plate of a series of shadows of varying density representing the various structures through which the rays have passed. The correctness of the diagnosis depends entirely upon the skill with which these various shadows are separated and interpreted. To interpret these shadows correctly one must know not only the appearance of the normal structure, but also the alterations that take place when there is a pathological process present. It therefore follows that the ability of the roentgenologist depends not so much upon his technical skill as upon his familiarity with the general problems of medicine and surgery.

When there is a variation from the normal he must know whether it is a developmental process or the result of disease. The bone of a child differs materially from that of an adult. In the former case it is in the growing stage, and too often a diagnosis of fracture has been made when the supposed crack

was the normal epiphyseal line. A thorough knowledge of the anatomy and development of the bone would have obviated this mistake. The reverse of this has occurred where a linear crack was present just below the head of a bone in an adult and was interpreted as the epiphyseal line. The thinning out of the cortex on the internal sides of the radius and ulna for the attachment of the interosseus membrane has often been mistaken for a periostitis. So it may be said that the first requisite of a good roentgenologist is a thorough knowledge of the normal anatomy, not only its appearance but also its normal development.

The roentgenologist should also be so thoroughly acquainted with every phase of the development of the bone that he can tell the approximate age of the individual by its appearance. If the epiphysis of the lower end of the radius has united at fourteen years, or is still present at twenty-five, it may possibly give the clue which will solve a very obscure case. Going a step further, the roentgenologist must know the constituent parts of the bones, their make-up and what rôle they play in the normal. He must be thoroughly familiar with the blood and lymph supply, as that shows how metastatic infection or malignancy reaches certain portions of the bone, and why other portions are omitted. In the same way he applies his knowledge to the joints, knows their constituent parts and what rôle each part plays. If he knows the blood and lymph supply of the cartilage he will know why in cartilaginous lesions the infection always starts from the periphery and never from the center of the cartilaginous mass.

After becoming thoroughly acquainted with the histology of the normal bone and the functions of its various parts, the roentgenologist must turn to the pathological side and know the changes that take place. He must know not only the intimate structure of the lesion, but also its method of progression. This does not occur at random, but follows well-defined laws based on the character of the lesion and on the roads of transportation. Having learned the normal and pathological

conditions of bone, how is he to translate them into what may be termed *x*-ray gross histology and pathology, for after all if he cannot connect the lesion with the *x*-ray plate, his knowledge is of no avail. It is in bridging the gap between these lesions and their shadows cast upon the plate that the expert knowledge of the roentgenologist comes into play.

Only two pathological processes are registered upon the plate; bone destruction and bone reproduction. The consideration of how and when these processes occur, where they are situated, and their relation to each other, enables one to make a diagnosis. Bone destruction may arise from some infection, or from a malignant growth. In either case what may be termed an irritant substance is introduced into the bone. This acts as an exciting agent, and, if it is an infection, more or less destruction of bone ensues, depending upon its virulence. If the irritant is of a benign nature it stimulates, and there is new bone formation around the substance, as this is nature's way of taking care of a pathological condition. The question will present itself as to why in one case there is destruction and in the other bone production. The point of contact between the lesion and the bone is the point of stimulation, and it takes nature some little time to lay down bone; so if the infection or tumor is virulent the point of contact is swept away and a new one formed before there has been time for new bone formation, hence the process is entirely destructive. On the other hand, if the point of contact remains stationary for a short period then nature has time to lay down new bone. So there are two well-defined changes taking place in bone, namely, destruction, indicating an advancing lesion, and production, indicating a lesion that is retrograding and probably under control.

In some processes there is continued destruction and no bone production, indicating that the lesion is a rapidly advancing one and out of control. This is seen in malignant tumors, such as carcinoma, round and spindle-cell sarcoma and virulent infections. The nature of the lesion determines the character of the destruction. An osteomyelitic infection spreads by

means of the Haversian canals and their intersecting ramifications, and thus may break out in a point distant from the original focus, leaving normal bone between. On the other hand growths advance by direct extension, and so fresh foci are not found separated from the parent growth with normal bone between. The character of the destruction, therefore, makes it possible to establish one point of differential diagnosis.

The production of bone at the junction of the lesion and the normal bone has already been mentioned. When new bone, then, is laid down at the edge of the destroyed area it is indicative either of an infection or of a benign growth. On the other hand new bone is sometmes seen within the lesion itself where there is normally no tissue that will produce bone. Such a condition probably indicates a bone tumor such as an osteoma or an osteosarcoma. Thus a second point of differential diagnosis is established.

When the lesion is in the medullary canal there is no bone production, but vacuolated areas appear in benign growths and infections, and the path of least resistance is taken, namely, up and down the canal, while malignant tumors grow in a spherical manner and tend to destroy the cortex by pressure. So a third point of differentiation is established—pressure effects, and the manner in which the lesion extends.

The above points may be termed direct evidence.

Then the remote effects must be considered, whereby changes occur in the bones, not directly due to the lesion, but rather to mechanical causes.

When some process, especially an infection, involves the bone, there is generally pain with consequent inability or lack of desire to use the particular part affected on account of discomfort. This disuse brings about a condition known as atrophy, which is not due to disease, but is entirely a disuse process. It is seen even in a normal bone if the part is put at rest. The calcium salts are absorbed and a very porous bone results. Each constituent part is present, but in much dimin-

ished quantity. This process is entirely functional, and the bone will quickly return to normal upon use. It is never a pathological condition.

At times there is a local atrophy following a pathological process. In fractures, where the blood vessels are destroyed, there is sometimes actual necrosis of the bone due to lack of nourishment. Atrophy, then, is an indication of disuse of the part, and thus calls attention to the fact that some cause must be found for its presence. As an example of this, an examination of the hip was requested in a certain case. The plate showed no lesion in the joint, but that portion of the femur shown upon the plate disclosed marked atrophy. There was no lesion present that would account for this condition. In discussing the case with the surgeon attention was called to this point, and the statement was made that for some cause unknown to the roentgenologist the femur must have been at complete rest for a long period of time. This elicited the information that there had been fracture of the lower third of the femur, and that both femur and pelvis had been in a cast for nearly twelve weeks. Atrophy, therefore, is a functional disuse process, and its presence must be accounted for, as it always means non-function frequently due to disease. On the other hand a knowledge that atrophy is absent is often of the greatest importance. In a Charcot joint the plate shows swelling and extensive destruction. In any other condition the joint would be painful and the part could not be used; but the lack of atrophy indicates that the joint was used, and hence there could have been no pain. Thus, by simple logic the absence of pain is deduced in a badly disorganized joint by the fact of the absence of atrophy, and a diagnosis is established.

So far the changes seen upon an *x*-ray plate have been discussed, and they may be termed direct evidence. Are there any other factors that may be obtained which will aid in a diagnosis? There are two such factors, and even these if necessary may be deduced from the plate by a skilled roentgen-

ologist. However, these factors, namely, sex and age, will be considered as indirect evidence.

Certain lesions peculiar to the female are seen but infrequently in the males. As an example, separation of the symphysis pubis which is common in the female after childbirth, is but rarely seen in the male except following severe trauma. Certain fractures are much more common in the male. Certain metastatic malignancies have a different bone distribution in the female than in the male.

Again aneurysms are relatively uncommon in the female. Consequently destruction of the vertebrae due to pressure from beneath is not often seen; destruction of the thoracic vertebrae in a female, therefore, is apt to be malignant, while in the male in the same region it may be a pressure result.

Age has a very important bearing. The bones differ somewhat at different ages, and the resulting injuries from trauma will vary according to age. As an example, take the same degree of injury to a hip and see what follows at different age periods. In the young an epiphyseal separation of the head of the femur results; between twenty and forty years a dislocation is produced; and after forty a fracture of the neck is sustained.

In the same way, if there is a malignant growth in the bone of a child it is certain that it is not a carcinoma, as that is an old-age disease. A joint infection in early youth cannot be a hypertrophic arthritis, as that, too, is an old-age lesion. Rickets and scurvy are seen only in the first few years of life. So laws of probabilities as to sex and age can be established. A law of probability also may be determined from the portion of the bone in which the lesion is situated, thus carcinoma is generally found in the middle of the shaft and sarcoma at the ends. These probabilities will be dealt with in the succeeding chapters.

When all the data that can be determined from the plate are obtained, it is well to write down every pathological

condition that can affect the part in question. Many can be eliminated immediately, but some few will still remain. In each of these there will be some factor that does not fit in with the plate, and finally, by exclusion, the list of pathological conditions will be narrowed down to one. Then the process must be reversed, and every shadow upon the plate must coincide with the lesion; if it does not, there has been an error which must be sought out. It is only when they agree that we may feel reasonably sure of the correctness of the diagnosis.

As has been stated, roentgenology is not a picture process, but a medical procedure based upon careful analysis and logical deductions from the shadows observed upon an *x*-ray plate and translated into pathological terms. This means—and it cannot be too strongly emphasized—that the skill of a roentgenologist will vary directly with his medical knowledge; and the value of the roentgenologist to the medical profession will be based upon this fact and not upon his technical ability. It must also be borne in mind that roentgenology is but one of many diagnostic methods, and that it has not as yet reached a stage of such precision that it may be considered infallible.

It is only by close cooperation with his colleagues, in conjunction with his medical knowledge, that the roentgenologist can advance his specialty. He has four friends who are only too willing to cooperate with him, and the more intimately he associates himself with them the better will be his progress. These four friends, the anatomist, the pathologist, the internist and the surgeon, the roentgenologist should most zealously cherish and esteem.

CHAPTER II

NORMAL BONES

CHAPTER II

Normal Bones

A NORMAL bone is composed of several highly organized structures having different functions. Some of these are plainly seen upon an *x*-ray plate, and others are demonstrable only when they are in what might be termed a pathological state. This is particularly true of such a structure as the periosteum. The constituent parts of the bone vary according to its particular type and also according to its function; for example, some bones have a shaft and cartilaginous articulating surfaces, as the femur or tibia; others, such as a carpal bone, have no shaft or periosteum, but are irregular in shape and are covered entirely by cartilage. While bones may vary in shape, yet they are composed of practically the same constituents modified according to the use to which nature intends them to be put.

The normal constituents are as follows:

The Periosteum. This is a fibrous sheath, rich in blood vessels, covering that portion of the bone not entering into an articulation. This structure has a very important function, as it is one of the bone producing elements, and its finely divided blood vessels penetrate the cortex, thus helping to nourish the bone. If the periosteum is torn from a bone we shall find the cortex studded with small bleeding points. In the young this sheath is quite thick and very vascular, but is loosely attached to the shaft and firmly adherent at the epiphyses, while in adults it is firmly attached to the shaft. This is important because when hemorrhage takes place beneath the periosteum in children it generally surrounds the entire shaft, as the periosteum is so loosely connected, while on account of the firm attachment in adults the hemorrhage is generally localized to one part.

In its normal state the periosteum casts no shadow upon the plate (Fig. 1), but in its pathological state there is invariably deposition of calcium salts (Fig. 2); consequently it can be easily demonstrated. Whenever the periosteum is visible upon a plate it indicates an abnormal condition.



FIG. 1.—A section of a normal radius and ulna. The periosteum cannot be seen as the calcium salts are not present in its normal state.

THE CORTEX. Beneath the periosteum we come to dense, hard bone, called the cortex, Fig. 3. This is composed of bone cells imbedded in masses of inorganic salts known as the matrix. This dense bone is pierced by numerous small Haversian canals which run parallel to the long axis of the bone and

are united by numerous intersecting canals. The canals play a very important rôle in certain diseases, as they may be termed the roads of transportation for the infections. At the ends of the bone the cortex expands into finely reticulated bone, rich in blood and lymph vessels, which is spoken of as

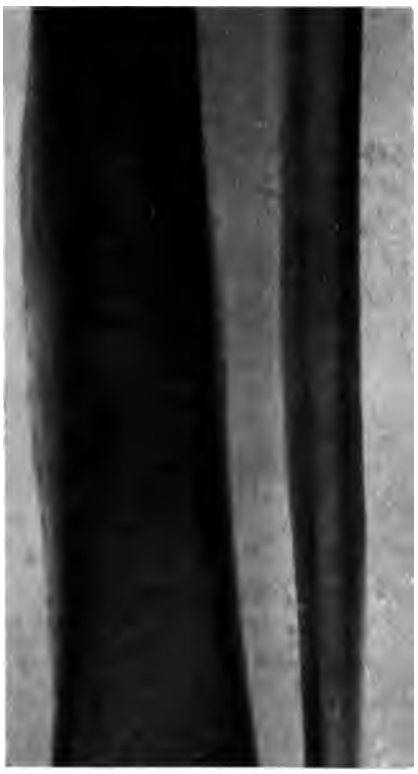


FIG. 2.—The deposition of calcium salts in the periosteum on the internal aspect of the tibia due to an inflammatory process of the periosteum.

cancellous bone, Fig. 4. These two types are simply variations in the amount of matrix present, there being more in compact bone and less in cancellous bone. The cortex and the cancellous bone are nourished partly by minute vessels from the periosteum and partly from the medullary artery.

THE MEDULLARY CANAL. Within a long bone there is a long narrow cylindrical channel known as the medullary canal (Fig. 5) containing the marrow fat, nerves, blood and lumph vessels. From an *x*-ray standpoint the medullary canal appears as a hollow channel, as its contained elements



FIG. 3.—Note the dense bony cortex of the tibia. In this area we have the bone cells, inorganic salts and the Haversian canals.

do not cast shadows. This canal does not traverse the entire bone, but merges at its ends into the cancellous heads.

THE NUTRIENT FORAMEN. At approximately the middle point of the shaft is the nutrient foramen, an aperture which allows the big vessels to enter the medullary canal. In some bones, such as the femur, there may be two such foramina. While this foramen cannot be demonstrated by the *x*-ray, yet it is of great importance, as it is the entrance by which metas-

tatic infections and malignancy gain admittance to the medullary canal.

CARTILAGE. The ends of the bones are covered by a hyaline material called cartilage. This is a dense, hard structure free from vessels. It varies in thickness, being thicker at its



FIG. 4.—There is no cortex present at the end of the bones; it and the medullary canal fuse and form a finely reticulated bony structure rich in blood vessels.

weight-bearing points, that is, its convex portion, and thinner at its concave portion. The ligaments of the joints are imbedded in it. When there is infection, the thinnest portion is destroyed first, therefore bone changes must be sought in those areas.

Joint cartilage does not undergo calcification, Fig. 6. It has poor recuperative power, and when calcification is present it means that the cartilage has been destroyed and replaced

by bone tissue. In other types of cartilage, such as the costal cartilage (Fig. 7), calcification is a normal change due to age but this never takes place in a joint. As cartilage does not contain blood or lymph vessels, metastatic infections and malignancy are not found in this tissue.



FIG. 5.—Within the center of the bone there is the medullary canal containing the nerves, blood and lymph vessels, and the marrow fat. This canal appears as a light channel upon the print.

Variations in bone architecture are due to differences in combinations of compact and cancellous tissues and the mechanical distribution of cartilage. We can, therefore, classify the bones as follows:

1. Long bones.



FIG. 6.—The space between the bones is filled with cartilage which casts no shadow. This type of cartilage does not undergo calcification with age.



FIG. 7.—This shows a different type of cartilage present in the costal cartilages which normally undergo calcification with advancing age.

2. Flat bones.
3. Irregular bones.

The long bones (Fig. 8) are made up of all the structures just

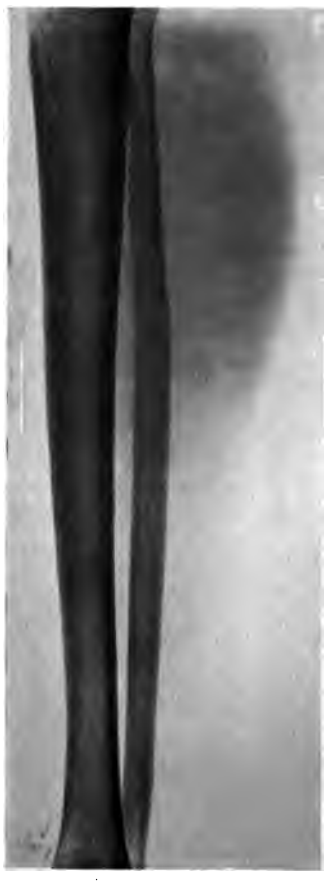


FIG. 8.—Tibia and fibula, showing cortex, medullary canal and cancellous bone.



FIG. 9.—A flat bone showing its cancellous structure, and, incidentally, the grooves for the blood vessels.



FIG. 10.—Carpal bone composed entirely of cancellous tissue with a very thin cortex. This bone comes under the head of what might be termed the irregular bones.

described, and may be the seat of fractures and disease not only of the shaft, but also of the joints, so both bone and joint lesions may be present.

The flat bones (Fig. 9) have slightly different architecture. There is no medullary canal, but the bone is made up of cancellous tissue lying between two thick plates of compact bone. There may or may not be joint cartilage present. Examples of this type are the innominate bones, scapulae, ribs, and the cranial bones. The first two, having articular cartilage, may be the seat of either bone or joint lesions, while the latter two are only subject to bone lesions.

The irregular bones (Fig. 10) such as the carpals and tarsals, are entirely cancellous and have a cortex of a very thin compact layer of bone; some of them are entirely covered with cartilage, as the carpal bones, or are partially covered, as the os calcis. So with some of the bones, those covered entirely with cartilage, only those lesions occur which may be termed joint diseases, while others, as the os calcis, are subject to both bone and joint diseases.

The vertebra may also be looked upon as an irregular bone; the upper and lower borders are covered with cartilage, and the sides with periosteum; so here also both bone and joint lesions may be present.

JOINTS. In a joint, which may be defined as the junction of two bones, a totally different condition prevails. No periosteum is present, but there are more or less large articulating cartilaginous surfaces and a capsular covering enclosing the joint known as the synovial membrane. The synovial membrane is a fibrous tissue sheath, but, unlike the periosteum, is incapable of producing bone. In a joint there are no normal elements which will produce bone. Therefore whenever bone is formed in a joint it is the result of destruction of cartilage and exposure of raw bone which then proliferates. Nearly all the elements of a normal bone are directly demonstrable, while the elements of the normal joint cannot be seen. In bone there is direct evidence of destruction, while in joints only indirect evidence is obtained, and the destruction must go on until the bone beneath is involved before direct evidence appears. As has been stated, cartilage casts no shadow; however, the space

between two joint surfaces is filled with cartilage (Fig. 11) and when this space is narrowed (Fig. 12) it shows that cartilage has been partially destroyed; thus we have what is termed indirect evidence of cartilage destruction. When cartilage is involved it is by direct contact either from the joint space or from the bone beneath. The lesion can never start from within the cartilage mass, as there are no vessels to carry in the infection.



FIG. 11. FIG. 12.

FIG. 11.—Normal space between the bones filled with cartilage.
FIG. 12.—Partial destruction of cartilage, indicated by narrowing of the joint space.

GROWTH. Thus far the discussion has been limited to a bone that has reached its full growth. Up to the age of epiphyseal union the bones differ very materially. Since bones have to grow, as does other tissue, and as they are rigid, fixed structures, some mechanism must be present that will allow them not only to lengthen, but also to expand. The expansion is very simple, being due largely to bone being laid down by the periosteum. This is done so insidiously that it cannot be dem-

onstrated. It may be likened to the constant replacement of skin epithelium.

To allow bones to lengthen nature has placed at each end of nearly all of them a very highly organized mechanism known as the epiphysis. When the bones first appear in fetal life they are composed entirely of cartilage, but soon centers of ossification appear and the cartilage is gradually transformed into bone. At birth all the long bones are fully developed, except as



FIG. 13.



FIG. 14.

FIG. 13.—A knee joint at a very early age, showing center of ossification for the lower end of the femur. The upper end of the tibia is composed entirely of cartilage, the ossified center not having appeared. Note the enormous width of joint space, composed entirely of cartilage, which casts no shadow.

FIG. 14.—The wrist of a child of seven showing the wide epiphyseal lines. Note particularly the epiphyseal line of the first metacarpal and compare with Fig. 15.

to size, with the exception of each end, and there the heads are composed entirely of cartilage, Fig. 13. At various ages, centers of ossification in the bone heads appear (Fig. 14) and increase in size, but do not unite until full growth has finally been attained. There is consequently a point where the

bone and ossified heads are united, but it is by a cartilaginous band which is wide in the young and becomes narrower with age, Fig. 15. It finally disappears by union with the shaft when full growth is attained, and under normal conditions the growth then ceases. This band of cartilage lays down new bone which causes the shaft to lengthen, and at the same time reproduces its own tissue. When it ceases to reproduce its own tissue then union takes place. This band is known as the epi-



FIG. 15.—A child of eleven years. The epiphyseal line of the first metacarpal has become quite narrow as this epiphysis unites at fourteen. The epiphyseal line of the radius is still quite distinct as it does not unite until eighteen.

physeal line, and is easily demonstrable by the *x*-ray. Any interference at this epiphyseal line, either by disease or injury, is attended with serious consequences to the particular bone involved. There are certain diseases in which the epiphyses are subject to either excess or retarded growth, and profound changes are invariably the result of these conditions.

JOINT LESIONS. While the bone is growing it always retains its mechanical shape, and thus lesions up to puberty are similar to those of adults. The joint lesions, however, are more

difficult to discern, as the heads are composed largely of cartilage which does not cast a shadow and consequently there may be various lesions which cannot be recognized by direct *x*-ray evidence. In an adult a fracture of the head of the humerus can be readily demonstrated, while in a newborn child, the head, being composed of cartilage (Fig. 16), may be broken or pulled off completely and yet not be recognized. It is the writers' belief that many of the so-called congenital abnor-



FIG, 16.—The head of the humerus of a newborn child. The entire head is composed of cartilage and may be injured and yet not be recognized by means of an *x*-ray examination.

malities are in reality injuries to the cartilage after birth, and that the resulting deformity is due to faulty growth or malposition of the cartilaginous fragment. Complete displacement of an epiphysis results in non-growth of that portion of the bone. These epiphyseal injuries are so often unrecognizable both clinically and roentgenologically at the time of injury that when later in life such a condition is seen it may be considered as congenital in origin. It may be stated almost as an axiom that before joint lesions in children can be demon-

strated by means of the *x*-ray they must always be more advanced than in adults.

Thus far it has been shown that in normal bones and joints there are definite variations between those of children and of adults, and that these variations may materially influence and aid in bringing about pathological conditions. The question



FIG. 17.—The shoulder joint in a man of fifty, showing atrophy from age. In this condition the bones are quite brittle, and fracture easily.

naturally arises whether there are any variations after the bones and joints have attained their full growth. The variations in youth can be classed as developmental. In adults there are no such changes; the bones have ceased growing. However, in adults there are alterations in some of the constituent parts. It may be roughly stated that the bones reach their full growth at twenty, and that from this age up to forty no change of any character can be noted. This is our most vigorous

period of life and our bones should be at their best. After forty we begin to slow up somewhat and this can be noted in our bones. Certain changes begin to take place, which may be termed retrograde, very slight at forty, but increasing with age. Up to forty our bones are dense, due to the inorganic salts, flexible and more resistent to stress and strain. After forty they gradually lose their flexibility and become somewhat brittle, Fig. 17. In children green-stick fractures are common owing to the flexibility of the bones; but in old age the fractures are always complete and sometimes comminuted as the flexibility has disappeared, leaving a more brittle bone. This is due to a gradual absorption of lime salts, so that the bone becomes more atrophic and offers less resistance to injuries and diseases. This atrophic condition is readily recognized upon an *x*-ray plate. There are certain types of fractures occurring after the age of forty which are extremely rare below that age. Thus the life of the bone may be regarded as having three distinct phases:

1. Up to the age of twenty; the stage of progressive development.

2. From twenty to forty; the stage of greatest development and strength.

3. From forty on; the stage of retrogression and weakening of the bone structures.

Effect of Sex. The second question to be considered is whether there are any changes in the structure of the bones in the male that make them differ from those of the female. The bones of both sexes are identical as far as structure is concerned, except that as a rule those of the female are shorter and narrower; but in proportion to their size, they are as strong as those of the male. The arrangement of the bones may be slightly modified in the female to accommodate them to functions peculiar to that sex. The pelvis (Fig. 18) will demonstrate this point. In the female the sacrum occupies a high position, so that its top plane is on a level or just slightly below a plane passed through the crests of the ilia. This causes a



FIG. 18.—The pelvis of a female. Note the high position of the sacrum with the corresponding position of the fifth lumbar in relation to the crest of the ilia.



FIG. 19.—The deep-set sacrum of a male. Again note the position of the fifth lumbar in relation to the ilia.

flaring out of the iliac bones and gives a wider and deeper pelvic canal, which in turn slightly alters the angle that the femur makes with the pelvis. In the male (Fig. 19) the plane of the iliac crests passes through the middle or top of the fifth lumbar vertebra, resulting in a narrow pelvis.

It is reasonable then to conclude that age and sex have a definite relationship to normal bones, and that they also have an effect upon the bone lesions when they occur.

In the following chapters injuries and diseases will be studied from the standpoint of sex and of the three age periods that have been enumerated.

CHAPTER III

EPIPHYSES

CHAPTER III

Epiphyses

In roentgenological diagnosis it is very important to have a thorough knowledge of the epiphyses of the bones. This portion of the bone plays such an important part in the growth and is so easily affected by injury and disease, that all the normal changes which occur up to the time of its union with the shaft should be thoroughly understood. It must be known therefore, at what time the center of ossification appears, the appearance of the epiphysis at various ages, and finally, the period when it unites and becomes an integral part of the bone. The age of a child of average development can be approximately determined by the absence of the centers of ossification or by the size of the ossified centers when present. This is very important diagnostically as some injuries and diseases are limited to certain ages.

It must be remembered, however, that the development of the epiphysis is somewhat variable, being influenced by a number of conditions. Nutrition is probably one of the greatest factors. It has been the general experience that in undeveloped and poorly nourished children, the growth of the centers of ossification is very materially retarded. It must also be borne in mind that the epiphyses in the newborn are, for the most part, composed entirely of cartilage, and since cartilage does not cast a shadow it cannot be demonstrated by an *x*-ray examination, Fig. 20. It is only when the center of ossification appears that information can be obtained, Fig. 21. We then judge of its size, position and contour to determine whether it is normal, pathological, or has been the seat of an injury.

It has been noted that malnutrition retards the development of the epiphysis, and that in the normal individual, there is a wide variation in the time of appearance of the centers of



FIG. 20. FIG. 21. FIG. 22.

FIG. 20.—A child of eighteen months with only two centers of the carpal bones showing.

FIG. 21.—A child of three years showing three centers of ossification of the carpal bones.

FIG. 22.—A child of ten years with delayed ossification. None of the carpal bones are developed as fully as they should be at this age.



FIG. 23.—A child of fifteen years with the center of ossification of the epiphysis of the acromion just appearing. Sometimes mistaken for a fracture.

ossification (Fig. 22) and the time of fusion. For practical purposes only those centers of ossifications which appear after birth need be considered.

SCAPULA. The scapula has seven centers of ossification of which but three need be mentioned here. They are the center for the coracoid process, the center for the outer end of the acromial process, and that for the inferior angle of the body of the scapula. The coracoid center appears at the end of the



FIG. 24.—A child of four years, showing separate centers of ossification for the head of the humerus. These fuse into one solid head at about the sixth year.

first year and unites at about the fifteenth year. The outer end of the acromion (Fig. 23) has one and sometimes two centers appearing about the fifteenth year and fusing about the eighteenth. The center for the inferior angle appears at the age of fifteen and fuses at about eighteen. *These epiphyses are sometimes mistaken for fractures.*

CLAVICLE. The sternal end of the clavicle has a separate center which appears from the fifteenth to the seventeenth

year and fuses at the twenty-third to the twenty-fifth. This epiphysis is seldom of pathological interest, but is a good index in determining the age of a patient.

HUMERUS. The upper end of the humerus has three centers: the head, appearing at the sixth or eighth month; and the greater and lesser tuberosities, appearing from the third to the fourth year, Fig. 24. These unite at about the



FIG. 25.—The epiphyses around the elbow joint at the fourteenth year.

sixth year into one big epiphysis. This epiphysis is of great importance, as it is a common seat of injury and disease.

The lower end of the humerus has four centers (Fig. 25); (1) the capitellum, appearing at one year; (2) the internal condyle, at five; (3) the trochlea, at ten to eleven; and (4) the external condyle, at twelve to fourteen. They remain as separate centers until the age of sixteen to seventeen, and then unite as a mass and fuse at the eighteenth or nineteenth year. The lower epiphysis figures very largely in injuries at the elbow joint in the first ten years of life.



FIG. 26.—The lower epiphysis of the radius. This epiphysis is a very important one as it is so frequently torn off and displaced backward.



FIG. 27.—The epiphysis of the olecranon at the fifteenth year. This is important as it is occasionally torn off.

RADIUS. The center for the head of the radius appears about the fifth and unites at the sixteenth to seventeenth year. This epiphysis is seldom injured. The lower epiphysis appears about the second year and unites at the seventeenth



FIG. 28.



FIG. 29.



FIG. 30.

FIG. 28.—A child of four years with four centers of ossification of the carpal bones.

FIG. 29.—A child of seven years with seven centers of ossification of the carpal bones.

FIG. 30—A child of eleven years. The center of ossification of the pisiform is now present, appearing between the eighth and eleventh year.

or eighteenth. This is also a very important epiphysis, as it is so frequently dislocated, Fig. 26.

OLECRANON. The olecranon center appears at eight or nine and fuses at seventeen. It is subject to frequent injuries, Fig. 27. The lower epiphysis appears at the fourth and fuses at the eighteenth year and is seldom injured.

CARPAL BONES. The carpal bone centers appear in the following order: os magnum, unciform, cuneiform, semilunar, trapezium, scaphoid and trapezoid; and a good working rule is that they appear one for each year in the order named above (Figs. 28 and 29), the center for the pisiform appearing between the eighth and eleventh year, Fig. 30. The metacarpal centers appear about the third and fuse at the seventeenth to eighteenth year.

PHALANGES. The phalangeal centers also appear about the third year and fuse at the sixteenth or seventeenth.

PUBIS AND ISCHIUM. The pubis and ischium unite at

from seven to nine (Fig. 31) and the acetabulum fuses at fifteen to sixteen. The center for the crest of the ilium appears at from fifteen to eighteen years and fuses at twenty-three to twenty-five.



FIG. 31.—The pubis and ischium unite at from seven to nine years. This plate of a child of five shows that complete ossification has not taken place.

The centers for the ischial tuberosity, the iliac spine and tubercle of the pubes, appear at fifteen and unite at twenty and are of little pathological importance.

FEMUR. The center for the head of the femur (Fig. 32) appears at the first year and fuses at seventeen to eighteen. The epiphysis is of great importance, as it is frequently dislocated.

The greater trochanter appears at the fourth and unites at

the eighteenth year. The lesser trochanter appears at the eleventh to thirteenth year and unites at seventeen. The center for the lower epiphysis of the femur appears at birth (Fig. 33) and unites at eighteen to twenty years. This epiphysis is important, as it is sometimes dislocated.

PATELLA. The patella center appears at the third to fourth year, Fig. 34.



FIG. 32.—A child of nine years showing the epiphysis of the greater trochanter. The lesser trochanter is not present as the ossified center does not appear until the eleventh year.

TIBIA AND FIBULA. The center for the upper epiphysis of the tibia (Fig. 35) appears in the latter half of the first year and unites at eighteen or twenty and is but rarely separated. The lower center appears at about two and unites at seventeen or eighteen years and is subject to injuries. The upper center for the fibula appears at three to four and unites at eighteen to

twenty-five years. The lower epiphysis appears at two and unites at seventeen to eighteen.

The centers for the os calcis, astragalus and cuboid appear before birth (Fig. 36); the external cuneiform at one year; internal cuneiform at the third year; the middle cuneiform and scaphoid at the fourth year. The separate center of the posterior extremity of the os calcis appears at the tenth and unites



FIG. 33.—A child under six months, showing the center of the epiphysis of the lower end of the femur which is present at birth. The center for the upper end of the tibia is not present as it does not appear until the last six months of the first year.

at the eighteenth year. The centers for the metacarpals and phalanges vary in their appearance from three to seven years and unite at about seventeen.

For practical purposes, it may be accepted that with the exception of the head of the fibula, which fuses after twenty years, union of the epiphyses of the long bones takes place approximately at eighteen years.

VERTEBRAE. Vertebrae (Fig. 37) arise from three centers, one for the body and one for each lamina. These are almost fully developed at birth; in the first year the laminae unite and at the third year the body and the arch join. At the sixteenth year secondary centers appear, one for the tip of each transverse process and one for the spinous process. At twenty-one a

thin circular plate of epiphyseal bone forms in the cartilage between the vertebrae, one above and one below each vertebra and unites between the twenty-fifth and thirtieth year. These epiphyses seldom play any part in injuries, but by variations in ossification they produce abnormalities, as in sacralization of the fifth lumbar vertebra, where there has been over-



FIG. 34.



FIG. 35.

FIG. 34.—The center for the patella appears at the fourth year. The age of this patient is about nine years and the patella has not reached its full size.

FIG. 35.—The well-developed head of the tibia at eight years.

development of one of the lateral masses. The reverse of this is seen where the lateral mass on one side of the fifth lumbar vertebra is under-developed and a scoliosis results.

These variations in the normal process of development and union of the epiphyses are important factors in the production of deformities. Certain diseases of infancy, notably rickets, lues and scurvy, affect the epiphyses, retarding growth and producing deformities. Then again, in cretinism and infantilism there is marked delay in the union of the epiphyses.

In cases of the latter condition all the epiphyses have been seen as late as the age of thirty. In the various types of chondrodystrophies the reverse takes place; the epiphyses unite at a much earlier age than normal, and consequently the bones are much shorter, but the periosteal growth continues until the bones reach their normal width.

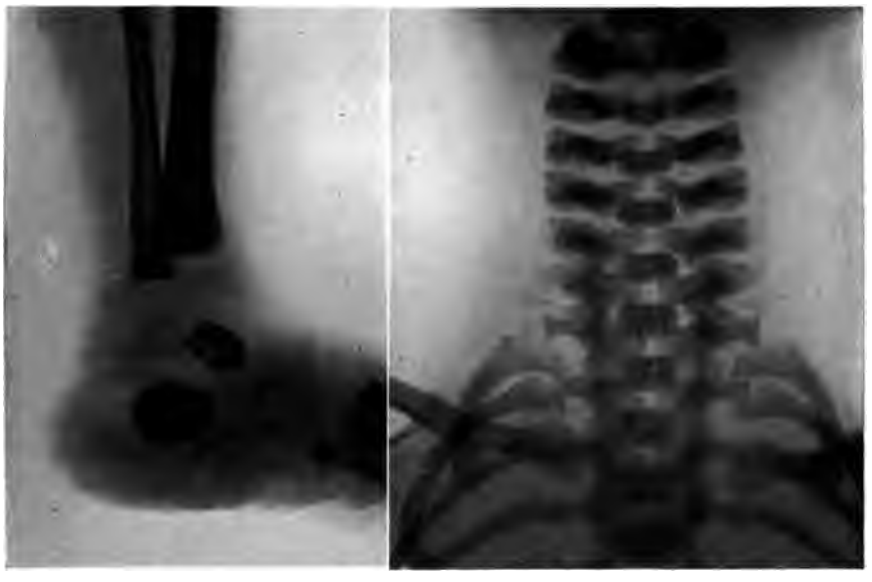


FIG. 36. FIG. 37.

FIG. 36.—Showing the centers of the ankle joint present at birth. The space between the leg bones and metatarsals is filled with cartilage.

FIG. 37.—Note the three centers present in the vertebrae, one for each lateral mass and one for the body.

There are certain variations as to the position of the epiphyses that must be borne in mind. The thumb metacarpal has its epiphysis at the proximal end, while the remaining four have their epiphyses at the distal end. This, taken into consideration with the fact that all the phalanges have their epiphyses at the proximal end and that the thumb has only two phalanges, suggests that the thumb metacarpal is in reality a phalanx, Fig. 38. This condition exists also in the big toe.

In a small percentage of cases the second metacarpal and the metatarsal, besides having epiphyses at the distal end, have additional ones at the proximal ends.



FIG. 38.—Showing the epiphysis of the first metacarpal at the base while the other metacarpals have their epiphyses at the distal end.

As has been noted, the epiphyses play an important part in injuries. The junction of the epiphysis and shaft is relatively weak and in injuries of that region epiphyseal separations are common. We seldom have dislocation, as the joint capsule is stronger than the epiphyseal union.

CHAPTER IV

FRACTURES OF THE UPPER EXTREMITIES

CHAPTER IV

Fractures of the Upper Extremities

A FRACTURE may be defined as a break in the bone texture or a solution of continuity. When such a condition takes place we have a pathological process in one or more of the component parts of the bone, directly dependent upon the severity of the trauma. The different types of bone have already been described. The character of fractures will vary with the type of bone involved, as transverse, oblique and spiral fractures of the long bones, or stellate fractures of the flat bones. The gravity of a fracture also varies not only as to displacement, but also as to its position. A fracture of a cranial bone is always serious whether displaced or not. The shaft of a long bone may not be perfectly reduced, yet the function and cosmetic results will be good. A perfect reduction of a fracture that extends into the joint may end in limitation of motion on account of callus protruding into the joint.

ELEMENTS AFFECTING FRACTURES

Muscular Tension. Two distinct elements enter into every fracture, trauma and muscular pull. Given a certain degree of trauma with the muscles relaxed, and no fracture may ensue, while with the same degree of trauma with the muscles in tension a fracture will occur. This is of importance, as in the latter condition a fracture may ensue from a very slight injury. In one case a condyle of the femur was broken off by suddenly attempting to cross the legs. Never rule out fracture because there is no history of trauma or only a slight one.

Every bone has a certain amount of flexibility, but when the muscles are under tension it becomes more or less rigidly

fixed, and a sharp blow will cause it to shatter, because it is held so rigidly that elasticity is lacking to take up the shock of the trauma.

AGE AND SEX. Besides the two main conditions mentioned above, another factor that enters into the production and type of fracture is age.

Up to the time of puberty the growing portions of the bones, the epiphyses, are but loosely united by bands of cartilage, which arrangement weakens the bone at those points. When the force of an injury centers at the epiphysis there is more apt to be an epiphyseal separation than a fracture. As an example of this it may be stated that an epiphyseal separation of the lower end of the radius is extremely common, while a fracture, such as a Colles's, is quite rare before puberty. It must be understood that we are referring only to ordinary trauma and not to fractures produced by crushing injuries or direct heavy blows.

While the bony structure before the age of puberty is most flexible, yet the presence of the epiphysis, as just mentioned, renders the bone as a whole relatively weak. After the epiphyses have united and up to forty years of age bones are probably at their strongest, and while in this period fractures are frequent, many sprains and dislocations are also seen which do not occur nearly so frequently in the age before puberty or in old age.

After forty bones begin to lose their flexibility, and there is a certain absorption of calcium salts, causing them to become brittle and fracture much more easily. In this period dislocations are not simple but are generally associated with fracture.

Certain fractures are met with in the old age period which are rarely seen in the first two periods. A fracture of the neck of the femur is an example of this. From a simple injury, such as a fall, it is extremely rare to see such a fracture result before forty years of age.

We also see many more fractures in the male than in the female. This is not due to structural weakness of the bones

but is the result of habits and customs. As a general rule a child is under the care of the nurse or mother up to five or six years, and accidents resulting in fracture may occur to either sex in that period. After that age and up to forty the male is engaged in more active sports and hazardous occupations which cause a great frequency of fractures. After forty years both sexes generally revert to the safe way of living and the occurrence of fractures is again distributed approximately equally between the two sexes. Age and sex bear a definite relationship to fractures.

Occupation. There are certain fractures which we may term occupational. Before the day of self-starters on automobiles, chauffeur's fracture of the radius due to cranking was not uncommon. This was a definite type of fracture and could generally be recognized as such. It was interesting to note as motors became more powerful how the fracture changed from a subperiosteal one without displacement to the displaced and comminuted type.

Condition of Soft Tissues. In addition to determining the presence of a fracture and the position of the fragments, the condition of the soft tissues must also be noted, and the presence or absence of atrophy and callus determined. A careful study of the plates will give this information.

Just after a fracture has occurred the soft parts surrounding the fracture will swell (Fig. 39) and this swelling will persist for about one week after the fracture has been immobilized. After that period the soft parts become normal in size, but as the immobilization continues atrophy of the soft parts also will begin to ensue, due to disuse. This is most marked in ununited fractures. Just as in bones this is a functional rather than a pathological process.

Bone Atrophy. When a fracture is present the affected part is held immobilized either by the application of splints, or if not treated the patient will hold the part immobile on account of pain. When a bone is immobilized disuse of that part results, and after a lapse of about three weeks the roent-

genogram will show a beginning porosity of the bone, Fig. 40. This gradually increases according to the length of time of immobilization. The condition is known as atrophy and is entirely a disuse process and not due to injury or disease, *per se*, so it also must be regarded as a functional rather than a





FIG. 39. FIG. 40.

FIG. 39.—Showing swelling of soft parts from a recent injury, resulting from a fracture of the lower third of the radius.

FIG. 40.—Old Colles's fracture where there has been marked limitation of motion with resulting atrophy of the bones. This marked degree of atrophy is never seen except with injury or disease of long standing.

pathological process. This can be readily demonstrated in a forearm where the radius has been fractured but the ulna is intact. At the end of five or six weeks the ulna will have become as atrophic as the broken radius.

Care must be taken not to confuse this general atrophy with the localized atrophy present at the ends of broken bones.

This localized atrophy is a true bone absorption and is the result of the tearing of the blood vessels with a resulting death of the bone nourished by the particular vessel injured.

With increasing age there is a gradual absorption of lime salts, producing a certain degree of atrophy of all the bones. This can be readily recognized, as all the bones will be equally affected, and the atrophy will not be confined to the injured bone.

Some writers use the phrase "atrophy of quality" when there is absorption of lime salts and the bone is more porous, and "atrophy of quantity" when the bone is smaller in size than normal. Atrophy of quantity, is a very misleading term, since it implies that an actual reduction in the size of the bone has taken place, whereas the difference in size is due to non-development and not to shrinkage. The undeveloped femur in a congenital dislocation illustrates this point.

When a bone is immobilized, atrophy begins to show in about three weeks and increases with the time of immobilization. In an old ununited fracture the atrophy may be so excessive that it is often difficult to get a roentgenogram which will properly demonstrate the bone.

Formation of Callus. It has already been mentioned that the periosteal covering of the bone in its normal state cannot be demonstrated upon a roentgenogram. When a trauma occurs to it, and especially if it is torn, the resulting reaction will be the gradual deposition of lime salts and the formation of new bone. This, taken in connection with the new bone thrown out at the end of the fragments, we speak of as callus, Fig. 41. In adults this lime deposition cannot be recognized as such until nearly four weeks have elapsed, but in children it may take place as early as the end of one week. Up to that period the bone may be firmly united, yet the *x*-ray will not show any calcium salts. During this pre-lime salt period we speak of the bone and periosteal reaction as soft callus. Callus is then first seen in about four weeks, and under ordinary conditions reaches its maximum in six weeks, after

which period it will be gradually reabsorbed. The amount of callus depends upon the severity of the injury to the parts and upon the amount of the displacement of the fragments. In the subperiosteal fracture with no displacement no excess callus will form—in fact, sometimes no callus is seen at all, while in a fracture with marked displacement there will be a



FIG. 41. FIG. 42.

FIG. 41.—Callus formation at the end of six weeks. Callus as a rule does not show much before the fourth week in adults.

FIG. 42.—Backward displacement of the lower end of the humerus. The periosteum was torn loose for a distance of two inches from the posterior aspect of the humerus. At the end of eight weeks the space between the torn periosteum and humerus has become completely filled with new bone.

great amount of callus. The greater the injury and the more the periosteum is torn the greater will be the new bone formation.

New bone formation does not take place at random, but is the direct result of stimulation. In all cases of fractures the stimulation is the injury, and consequently the new bone will be produced as far as the injury extends. This is the reason

that in some fractures, particularly in supracondyloid fractures of the lower end of the humerus, subperiosteal bone will be seen extending upwards a third or a half of the shaft, as in such a fracture the periosteum will be torn loose that far, Fig. 42.

If we take into consideration soft tissue swelling, atrophy and callus and their relation to each other we can relatively tell the age of the fracture. This is sometimes of great importance, as the following case will illustrate. A man of forty-five entered suit for $50,000 against a construction company for a fracture of the neck of the femur alleged to have been sustained while in the employ of the defendants. An *x*-ray examination made two days after the injury showed a fracture of the neck of the femur. At the trial the defendants called a roentgenologist who after seeing the roentgenograms of the broken bone was able to state that the fracture was at least six months or a year old. This opinion was rendered upon the following data obtained from the plate: There was an extreme degree of atrophy present, no callus formation but partial absorption of the neck of the femur. A temporary postponement of the case was granted. Further testimony obtained showed that the patient had fractured his hip several months previously while in the employ of some concern in the West and the defendants won the case.

To summarize briefly the points in determining the age of the fracture: In a fresh fracture there is soft tissue swelling, no atrophy, no callus. In a fracture of two weeks duration there is no swelling, no atrophy, no callus. In a fracture of four weeks duration there is slight atrophy of the soft tissues, atrophy of bone and beginning callus formation. In an old ununited fracture there is excessive atrophy of both soft tissues and bone, no callus formation and the edges of the fracture have become smooth and slightly eburnated.

Fractures from Pathological Causes. The above description applies only to traumatic injuries. In a certain class of cases fractures occur not as the result of trauma, but from

destruction of bone following some pathological process. This is particularly true in osteomyelitis and in malignant tumors of the bone and in benign conditons where the bone undergoes a rarifying process either local, as in bone cysts, or general, as in osteomalacia and osteitis deformans. In these conditions the fracture, position of fragments and method of repair will be influenced by the pathological process antedating the fracture. In all fractures it is well to bear in mind that the older the patient the more slowly repair takes place and the greater the chance of non-union.

The duty of a roentgenologist is not only to determine the presence or absence of a fracture but, if a fracture exists, whether there is displacement or not. By data obtained from the plate he should be able to determine the approximate age of the fracture, so that if no callus is present he can state whether it is too early for callus, or that there is delayed callus formation or the condition of non-union exists.

FRACTURES CLASSIFIED ACCORDING TO AGE

The foregoing description applies to fractures in general. An attempt will now be made to describe briefly the more common fractures that the roentgenologist encounters. These fractures will also be discussed from the standpoint of age periods, as follows:

1. Up to twenty, or the first age period.
2. From twenty to forty, or the second age period.
3. Over forty, or the third age period.

Fractures of the Head

THE SKULL. Being a semi-hollow sphere, the skull has a certain elasticity, and this in combination with the close fitting integuments, such as skin, muscle and periosteum on the outside, and the intracranial contents, gives rise to variations in its fractures. In other words, displaced fractures are not, as a rule, encountered, but cracks in the cranial bones; though,

of course, when there is a very severe blow, as from a rock or hammer, or from a fall on some blunt object, there will be depressed fractures (Fig. 43), which are easy to recognize. The linear fractures (Fig. 44) are often very difficult to detect and may be mistaken for the grooves in the bones which carry the blood vessels. The importance of detecting these linear fractures is not in locating the fracture itself, but because



FIG. 43.—V shaped fracture with depression in the frontal region.

its presence indicates a probable hemorrhage beneath it; and for this reason, on account of the delicate brain structure, it is very necessary to determine the location of such a fracture.

Fractures of the skull may be divided into those of the vertex and those of the base. Both types may come from direct violence or may be the result of transmitted force, so that in such injuries a careful study of all parts of the head

should be made irrespective of where the actual trauma occurred. Since these fractures are due to violence they may occur at any age, but are most common in both sexes in the first age period and in males in the second age period.

As linear fractures of the vertex may be present without a brain lesion and may be overlooked, many cases of con-



FIG. 44.—Linear fracture in the occipital region. This was only differentiated from the groove for a blood vessel by stereoscopic examination.

cussion are probably fractures of the vertex. Fractures of the base are always accompanied by grave symptoms and consequently are *x*-rayed, hence fracture in this location is not so often overlooked. Every injury of the head should be examined; if this were systematically done the recorded percentage of fractures would be materially increased.

The important thing to remember is that the majority of

fractures of the skull are not important in themselves, but are merely indicative of hemorrhage, pressure or injury to the delicate brain structure lying beneath.

FACIAL BONES. These fractures are due to direct violence, such as severe blows or a fall upon some blunt object. On account of the complexity of the *x*-ray shadows in this



FIG. 45.—Fracture of the malar bone with hemorrhage into the sinus.

region it is frequently difficult to make the diagnosis from the plate, Fig. 45. Fracture of the nasal bone when displaced may be easily recognized. The zygoma is next in frequency and may also be recognized. Fractures of the malar bone and superior maxilla, unless badly crushed, may be overlooked. These fractures are frequently associated with hemorrhage into the sinuses.

MANDIBLE. Fractures of this bone are very rare in early childhood, but do occur in the latter part of the first age



FIG. 46.—Fracture of the mandible through one of the molar sockets followed by osteomyelitis.



FIG. 47.—Fracture of the condyle of the mandible with displacement.

period. It is by far the most common of all fractures of the facial bones in the second age period. In the white race at least eighty per cent occur in the male. In the colored race only about sixty per cent occur in the male and this is not due to structural weakness, but to social conditions.

The common site of the fracture is in the ramus near the angle of the jaw, and the line of fracture is transverse or slightly oblique, Fig. 49. The nearer the fracture is to the angle the greater the chance of displacement. Since the fracture generally extends through one of the tooth sockets infection is common, and these fractures are frequently associated with osteomyelitis, Fig. 46.

Pathological fractures are quite common in this region as the result of infection, cysts or growths. Fractures of the condyle and coronoid process are rare, Figs. 47 and 48.

Fractures of the spine will be discussed separately in the chapter on the spine. (See Chapter XII.)

Fractures of the Upper Extremities

CLAVICLE. Fractures of the clavicle may be divided into:

1. Fractures of the inner third.
2. Fractures of the middle third.
3. Fractures of the outer third.

Fractures of the inner third are relatively infrequent and seem to occur by direct trauma, such as a severe blow. Fractures of the middle third, especially at the junction with the outer third, are the most common of all. Fractures of the outer third, especially near the acromial end, are also quite frequent.

In fracture of the inner third the few cases observed by the writers were all in the second age period. Fractures of the middle third are most common in childhood and decrease in frequency with the approach of old age. This is probably due not to structural weakness, but to the fact that the danger of trauma decreases with age. In the first age period



FIG. 48.—Fracture of the coronoid process of the mandible. The fragment extends down to the third molar.



FIG. 49.—Oblique fracture of the mandible, not complete.

these fractures of the middle third may be of the green-stick variety (Fig. 50) with marked bowing or angulation, or complete fractures with displacement. They may be either transverse or oblique. In the transverse type in children reduction seems to be more difficult than in the adult and the fragments frequently have to be wired. These fractures may result from very slight trauma and may be unrecognized. In one case of a child of two years a diagnosis of sarcoma was made. The *x*-ray examination showed a fracture, and the hard mass present was callus formation. In the second age period the middle third is frequently broken and is generally oblique



FIG. 50.—Green-stick fracture of the outer third of the clavicle in a child.

with the outer fragment displaced downward. In the third age period the middle third is less frequently broken than the outer third. In the young fracture of the outer third is common, and the fragment is generally displaced downwards, Fig. 51.

In the second and third age periods there is one fracture of the outer third that deserves especial attention, since the clinical diagnosis of this is often wrong. In these periods, dislocation upward of the clavicle at its acromial end is quite common. In a number of cases, however, instead of a true dislocation taking place, the tip of the clavicle is fractured, Fig. 52. It remains *in situ*, but the clavicle rides up and the prominent end that is uppermost is often mistaken for the

end of the clavicle when in reality it is the fractured end of the shaft.

SCAPULA. Fractures of the scapula, excepting the acromion, are relatively uncommon and many of them, especially of the body, are not recognized. They may be divided into fractures of the body, spine, glenoid fossa, coracoid lower angle, and acromion.

Fractures of the body (Fig. 53) generally occur in the subspinous fossa starting just below the glenoid fossa, and are



FIG. 51.—Fracture of the outer third of the clavicle with slight displacement.

often incomplete. Fractures of the spine (Fig. 54) are uncommon unless associated with fractures of the body. Fractures of the glenoid (Fig. 55) are quite rare, and generally the lower third is involved, though the writers have seen one case where the glenoid was separated from the body. Fractures of the coracoid (Fig. 56) are also uncommon; the tip is sometimes pulled off by muscular violence. The lower angle is occasionally broken off and may be displaced. Care must be taken not to confuse the epiphysis of the angle for a fracture. Fracture of



FIG. 52.—Fracture of the extreme tip of the clavicle, the small fragment remaining *in situ* while the shaft of the clavicle has ridden up, simulating a dislocation.



FIG. 53.—Fracture of the body of the scapula with the glenoid displaced inward.

the acromion (Fig. 57) is most common and the outer tip is frequently pulled off instead of an acromio-clavicular dislocation. Scapular fractures are uncommon in the first age period and most frequent in the second age period.

HUMERUS. In the first age period we have the epiphysis present, and this relatively weakens that portion of the arm, yet separation is very uncommon, Fig. 58. The writers have



FIG. 54.—Fracture at the base of the spine of the scapula.

had only two such cases. In these cases the head remained in the glenoid fossa but rotated slightly allowing the shaft to slip slightly upward and outward. In young infants this injury may occur more often than is supposed, but since no ossification centers are present it cannot be demonstrated, as cartilage does not cast a shadow upon the *x*-ray plate.

Upper End. The most common fracture in this age period is in the upper third of the shaft or in the surgical



FIG. 55.—Fracture and slight displacement just beneath the glenoid fossa.



FIG. 56.—Linear fracture at the base of the coracoid process.



FIG. 57.—Fracture of the tip of the acromial process with displacement.



FIG. 58.—Epiphyseal separation of the upper end of the humerus. Note the rotation of the head with riding up of the shaft.

neck, Fig. 59. These are generally transverse, and the upper fragment is displaced outward. Fracture of the greater tuberosity is uncommon.

In the second and third age periods fractures of the anatomical neck may occur. In the experience of the writers this is a very rare fracture; while the clinical diagnosis of this fracture



FIG. 59.—Fracture of the surgical neck of the humerus in a patient over fifty, associated with a fracture of the greater tuberosity.

is made, the diagnosis is seldom borne out by the *x*-ray findings. Only two such cases have come under our observation.

Fracture of the surgical neck of the humerus is the most common, most frequent after the age of twenty. This fracture may be transverse, slightly oblique or impacted. Displacement varies according to the nature and severity of the trauma. It

may be associated with fracture of the greater tuberosity or with dislocation of the head of the humerus.

In the second age period the fracture is confined to the surgical neck, and is seldom associated with fracture of the greater tuberosity or dislocation of the head. The shaft, when displaced, as is also the case in the first age period, is generally pushed to the inner side, Fig. 60.



FIG. 60.—Showing the outward displacement of the head of the humerus in a fracture near the surgical neck.

In the third age period this is by far the most common of all fractures of the humerus, and is frequently associated with fractures of the greater tuberosity (Fig. 61) or dislocation of the head. The capsule is often badly torn, so that the end of the shaft rides up into the glenoid fossa and displaces the head outward, Fig. 62. Where the head is not dislocated it rotates slightly outward, so that the fractured surface points outward. When the fracture unites in this position the arm can only be raised to a right angle. This is due to the fact that at the time of union the head is in a position of partial rotation and when the arm is raised to a right angle the head, on account of its abnormal position, is in complete rotation. Occasionally fracture of the surgical neck is accompanied by a "T" fracture through the head, and then the internal portion of the head is dislocated, lying generally beneath the coracoid or in the axilla.

Fracture of the tuberosity alone is quite common and is

often unrecognized, as there may be no displacement of the fragment, Fig. 63. It is only after there is marked limitation of motion that something more serious than a bruise is suspected. Occasionally the attachments of the scapular muscles will be torn loose with a small bony fragment. This small fragment will be just external to the greater tuberosity. Care must be



FIG. 61.—Old fracture of the greater tuberosity and surgical neck of the humerus with atrophy from disuse.

exercised not to confuse this condition with subdeltoid bursitis with a calcium deposit in the wall of the bursa. Sometimes it is impossible to differentiate the two conditions. The following case will illustrate this point.

A man, aged forty, fell striking his left shoulder. Two hours after the injury swelling became pronounced and was accompanied by severe pain and limitation of motion. The *x*-ray disclosed what was taken to be a small fragment of bone torn

loose from the tuberosity. The arm was immobilized but there was no relief from pain. There had been no past history of any discomfort in or injury to that shoulder. It was finally decided that the apparent fragment of bone was in reality a deposit of calcium in the bursa. Operation confirmed this and with removal of the bursa the patient made a complete recovery.



FIG. 62.—Fracture of the surgical neck of the humerus with the end of the shaft in the glenoid fossa and the head displaced outward.

While every injury, especially in elderly individuals, should be viewed with suspicion as to fracture, it must be borne in mind that there is always present a quiescent low-grade arthritic condition. This may never have troubled the patient, yet an injury to the part may cause this arthritis to flare up and produce such acute pain in the joint that a fracture will be suspected.

Shaft. Fractures of the shaft are common and may occur anywhere between the surgical neck and the supracondyloid ridge, though fracture of the middle portion is most frequent. There is no definite displacement, as this varies according to the nature and severity of the trauma. In the first age period (Fig. 65) green-stick and oblique fractures are more frequent, while in the second and third age periods, transverse and comminuted fractures are more common, Fig. 66. The one interesting thing in fractures of the shaft is that non-union is quite frequent, due partly to the displacement of the frag-

THE MEDICAL LIBRARY
ST. LOUIS UNIVERSITY
MEDICAL CENTER
ST. LOUIS, MO.

ments and partly to muscle or fasciae lying between the fragments.

Lower End. Fractures of the lower end of the humerus



FIG. 63.—Old fracture of the greater tuberosity of the humerus, not recognized at the time of injury.

below the supracondyloid ridges are the most common of all. They may be divided into:

1. Supracondyloid fractures (epiphyseal in the young).
2. Fracture of the external condyle.
3. Fractures of the internal condyle.
4. Fractures of the internal epicondyle.
5. Fractures of the external epicondyle.
6. Fractures of the capitellum and trochlea.

The supracondyloid fractures may be transverse or oblique, and when close to the condyles may extend through the olecranon

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305



FIG. 64.—Epiphyseal separation of the lower end of the humerus. The joint is carried back with the fragment.

fossa, and there is often present a "T" fracture extending into the joint. The lower fragment may be displaced laterally or posteriorly according to the line of the fracture. In the first age period the supracondyloid fracture generally involves the epiphyseal line, and in the very young the fragment is usually displaced backwards and slightly to the inner sides, Fig. 64. It is a very difficult fracture to reduce. The internal displace-



FIG. 65.—Green-stick fracture of the humerus in an infant with no displacement. Just discernible.



FIG. 66.—Spiral fracture of the humerus. Such fractures are more commonly seen in the first age period.

ment of the lower fragment can be corrected and held. The posterior displacement can be corrected, but generally cannot be held, Fig. 67. When this fracture occurs the periosteum on the posterior surface is torn loose for some distance along the shaft. In such cases where reduction cannot be obtained the fragment may be left in this position and the displaced periosteum will throw down a strong bony bridge. When this has taken place the lower end of the shaft will be anterior and will prevent flexion. An open operation in chisel-



FIG. 67.—An attempted reduction of a supracondylar fracture of the lower end of the humerus. This was reduced under fluoroscope, but the fragment could not be held in the plaster cast and reverted to the original displacement.

ing off this fragment will give a perfectly functioning elbow. This fracture is often diagnosed as dislocation of the elbow.

Fractures of the epicondyles in children often cannot be demonstrated on account of their being entirely cartilaginous. The internal epicondyle is most frequently fractured. The writers have never seen a fracture of the external epicondyle. Fractures of the condyle are most common in this age period, and fracture of the external condyle is the more common

of the two. They may or may not be displaced, varying according to the nature and severity of the trauma. In the second and third age periods the supracondyloid fracture generally takes place just where the shaft expands to form the condylar portion of the lower end. These fractures are oblique, and the fragment may be displaced forward or backward according to the nature of the injury. Fractures of the condyles (Fig. 68) are more common in the second age period than in the third. The supracondyloid fracture in the second age may be accom-



FIG. 68.—Fracture of the external condyle of the lower end of the humerus. The fracture almost extends into the fossa.

panied by a "T" fracture into the joint. The fragments may be more or less displaced.

Fractures of the capitellum and trochlea are quite rare except where there is a crushing injury of the joint.

In injuries of the lower end of the humerus in young children care must be exercised in making a diagnosis of "no fracture." We must bear in mind that a great portion of the joint is cartilaginous and that a fragment of cartilage may be broken off and yet not be demonstrable by the *x*-ray. In such cases it has always been the custom of the writers to return a diagnosis stating that no fracture can be demonstrated.

FOREARM. *Upper End.* Fractures of the olecranon may occur at any age, but seem to be most common in early adult

and middle life, though occasionally in the young the epiphyses may be torn loose. Besides trauma, muscular pull plays an important part in this fracture, the writers having seen such an instance from throwing a base ball.

The fracture may take place in any portion of the coronoid fossa and is very seldom comminuted. The position of the fragment depends entirely on whether the triceps attachments are torn loose. If attachments are intact there will be no separation (Fig. 69), but if torn, the fragments may be widely



FIG. 69.



FIG. 70.

FIG. 69.—Fracture of the olecranon process. There is no displacement as the fascia has not been torn.

FIG. 70.—Fracture of the olecranon process where the fascia has been torn which has allowed the fragment to separate and rotate.

separated, Fig. 70. It is interesting to note that the chance of fibrous instead of bony union increases with the nearness of the fracture to the tip of the olecranon; and, conversely, as the fracture approaches the shaft, bony union generally takes place.

A fracture of the coronoid process (Fig. 71) is generally associated with backward dislocation of the ulna. It is very uncommon as a simple fracture. The line of fracture is roughly parallel or slightly oblique to the long axis of the shaft. While

the writers have seen such cases they are extremely uncommon, and in none of the cases observed was there any displacement of the fragment.

Fractures of the head of the radius are frequent and may vary from cracks to comminuted fractures with displacement



FIG. 71.



FIG. 72.

FIG. 71.—Fracture of the coronoid process and the head of the radius with dislocation of the joint not completely reduced.

FIG. 72.—A small fragment of the head of the radius broken off. The small calcified areas above this fragment are in the ligaments and are probably due to calcification of small hemorrhages. Fracture is six months old.

of the fragments. This fracture is common in early adult life and old age. It is very uncommon in the young and the writers have never seen a case of epiphyseal separation.

Fractures of the radial head, unless displaced, are often overlooked. It is the writers' experience that careful examination of the radial head in injuries of the elbow will show fractures without displacement to be much more frequent than is commonly supposed, and that these fissure fractures are

seldom recognized at first. In fact the patient does not consult a surgeon at first, thinking he has only a bruise; but with no amelioration of discomfort, at the end of a week or ten days, he seeks professional advice and *x*-ray examination shows the fracture. The writers saw four such cases in one afternoon, and





FIG. 73. FIG. 74.

FIG. 73.—Fracture of the head of the radius with part of the head displaced.
FIG. 74.—Fracture of the neck of the radius with marked anterior displacement.

all had sustained their injuries a week or ten days previously. The fracture may be confined to just a part of the outer edge of the head (Fig. 72); it may extend into the shaft; or it may be comminuted (Fig. 73), and divide the head into several fragments. When there is no displacement and the fracture is healed, a large percentage of the cases do not get complete extension of the arm. In the comminuted type, where one or more fragments are displaced, non-union will sometimes take place, and in that case the fragment acts as a foreign body and

has to be removed. Sometimes when displaced they will unite with excess callus and interfere with rotation.

Fractures of the neck of the radius (Fig. 74) are much less frequent than those of the head. The line of fracture is



FIG. 75.



FIG. 76.

FIG. 75.—Lateral view showing fracture of the upper third of the ulna with anterior dislocation of the head of the radius.

FIG. 76.—Same condition as that shown in Fig. 75 from an antero-posterior position.

generally oblique, and there is usually displacement. It is often very difficult to reduce and hold these fragments in place. Displacement with non-union is fairly frequent.

Fracture of the ulna (Figs. 75 and 76) alone is generally seen in the upper third just behind the coronoid fossa and

is frequently associated with dislocation of the head of the radius forward, Fig. 77.

Shaft. Fractures of the shaft of both bones of the forearm are probably next in frequency to Colles's fracture, and may



FIG. 77.



FIG. 78.

FIG. 77.—Old fracture of the upper third of the ulna with forward dislocation of the radius. The radius has slipped well up on the head of the humerus, due to the shortening of the ulna from absorption.

FIG. 78.—Green-stick fracture of radius and ulna showing angulation but no displacement.

occur at any age period. Fractures of the middle and lower third are the common situations, and are relatively infrequent in the upper third. Unless the fractures are of the green-stick (Fig. 78) or subperiosteal variety invariably there is displacement with more or less over-riding of the fragments, Fig. 79. This displacement may be so excessive that union will not take place, or if it takes place the excess callus may actually

produce a bony synostosis and rotation will be lost. In children this fracture is often incomplete and of the greenstick variety.

Lower End. Fractures in this region may be advantageously studied according to the three age periods.

In the first period the most important part of the lower





FIG. 79. FIG. 80.

FIG. 79.—Fracture and displacement of radius and ulna.

FIG. 80.—Backward dislocation of the epiphysis of the lower end of the radius. The joint is carried back with the epiphysis.

end of the radius is the epiphysis. Its union with the shaft is relatively weak when compared with the shaft of the bone, and trauma localized at that point will frequently displace it (Fig. 80) instead of breaking the bone. When this takes place the condition is much more serious than that obtaining with an ordinary fracture. In a fracture with faulty reduction

the end result is a deformity; with a displaced epiphysis not only the deformity results but, what is infinitely more important, the growing portion of the bone is destroyed. If the epiphyseal separation is not reduced, the deformity steadily increases as the ulna continues to grow, while the end of the radius remains stationary. This will result in a marked deflection of the hand to the radial side, continuing to increase until the ulna has reached its full growth.



FIG. 81.—Epiphyseal separation of the lower end of the radius with a small fragment torn off from the radius.

This epiphyseal separation of the radius is sometimes accompanied by a chipping off of a small fragment of bone from the ulnar side of the diaphysis of the radius, Fig. 81. Fracture of the styloid of the ulna, as in Colles's fracture, is also occasionally associated with this separation; but the writers have never seen the ulnar epiphysis torn off. Occasionally after a trauma one will find a swollen wrist and limitation of motion; the *x*-ray examination, however, will disclose no separation, but the epiphyseal line seems to be a trifle wider and more irregular than is normally seen. This swelling and tenderness may persist for several weeks; and in one case under the writers' observation, without any additional injury the epiphysis became slightly displaced, though it was in normal position just after the injury. This gives rise to the belief that following trauma the epiphysis may partially slip off and then snap back into position again. When such a condition is suspected the arm should be placed in splints and treated as an ordinary fracture.

When the patient falls upon the palm of the hand the epiphysis is dislocated posteriorly; if the hand is flexed and the patient falls upon the dorsum then the dislocation is anterior, but this is extremely rare. If the trauma is sufficient to produce fracture and the epiphysis does not yield, then the radius





FIG. 82. FIG. 83.

FIG. 82.—Fracture of both bones of the forearm, just below the point where a Colles's fracture would occur in the third age period.

FIG. 83.—Fracture in the second age period where both bones of the forearm are broken, resulting from a fall which would have produced a Colles's fracture if the patient had been older.

fractures an inch to an inch and a half below the epiphysis (Fig. 82) and is frequently associated with a fracture of the ulna at the same point. When this condition takes place the fragments generally override, and on account of the pronator muscles it is often impossible to reduce them without an open operation.

Occasionally the ulna alone will be fractured in this same region. In this age period green-stick and subperiosteal fractures are frequent. On account of the flexibility of the bones in

this period it is unusual to see comminuted fractures resulting from simple injuries.

In the next age period the epiphyses of the bones have united, and trauma in this region will cause a fracture of the radius or ulna or both about an inch and a half below the joint,



FIG. 84. FIG. 85.

FIG. 84.—Colles's fracture with marked displacement backward of the lower fragment carrying the joint with it.

FIG. 85.—Colles's fracture with anterior displacement of the lower fragment due to falling on the dorsal instead of the anterior surface of the hand.

and as in the first age period we may have the same deformities, Fig. 83.

During this period Colles's fracture is unusual. In repeated series of consecutive Colles's fractures resulting from falls we have found that only from 1 to 2 per cent occur in this age

period. From direct blows, such as cranking a car, we see Colles's fractures; but this type we may term occupational.

In the last age period the Colles's fracture predominates. A Colles's fracture is essentially a fracture of the middle-aged and old. Repeated series of this fracture show that from 98 to 99 per cent are above the age of forty. This is no doubt due to what may be termed senile changes in the bone, *i.e.*, loss of



Fig. 86.



Fig. 87.

Fig. 86.—An old Colles's fracture with backward displacement, the usual position of the fragment.

Fig. 87.—Impacted Colles's fracture with no displacement. This type of fracture is frequently mistaken for a sprain.

flexibility and absorption of calcium salts, thus causing the bone to become brittle.

This fracture is through the cancellous portion of the end of the radius; it extends across the bone about three-quarters of an inch below the joint, and is generally associated with a fracture of the styloid of the ulna. In the writers' series of cases the sty-

loid of the ulna was broken in from 60 to 70 per cent of the cases.

This fracture may vary from simple to comminuted, and, as in epiphyseal separations, the lower fragment may be displaced posteriorly (Fig. 84) or anteriorly according to whether the fall was on the palmar or dorsal surface of the hand (Fig. 85), the former being by far the most common.

The roentgenologist probably sees more old Colles's fractures than any other type of fracture, and this is because such a fracture if improperly set will give more trouble than any other. In cases where bad end results are obtained it is due to one of the following three causes:

1. Posterior dislocations, where the fragment has not been reduced, Fig. 86.

2. Impaction, where the shaft is driven into the fragment without any displacement at all. In such cases on account of the good alignment of the bone and lack of displacement the fracture is frequently dressed without reduction, Fig. 87.



FIG. 88.—Old Colles's fracture where there is angulation without displacement. Note the divergence of the axes of the shaft and fragment.

3. Angulation, where the fragment has not been dislocated but has turned upon its axis, so that while there is no displacement, yet the long axis of the wrist and fragment does not correspond to the long axis of the radius, Fig. 88.

The vast majority of impacted fractures result in painful and partially stiff wrists. If such a fracture is examined three weeks after reduction there will be found absorption of the ends of the fragments, and with the muscular tension these fragments are pulled together and shortening is produced. If, however, the impaction is broken up, then the fragments are separated and the consequent hemorrhage taking place between them keeps the fragments apart, so that when union takes place



FIG. 89. FIG. 90.

FIG. 89.—A Barton's fracture which follows approximately the epiphyseal line if that were present. A very rare fracture.

FIG. 90.—An old fracture of the styloid of the radius. Note the marked atrophy from disuse.

there is no shortening. It is a safe rule to follow that where the condition of the patient permits, every impacted Colles's fracture should be broken up. If this is done the end results will be infinitely better.

The important point to remember is that it is the anterior or posterior dislocations which produce the more or less painful and partial limitations of movements, while lateral

displacement gives an enlarged wrist, but it is not painful nor does it limit motion.

In middle and old age every injury to the wrist must be looked upon as a probable fracture, as the so-called sprains in the third age period are very rare. This is particularly true of those with impaction, as there is no displacement and on account of lack of deformity they are frequently overlooked unless *x*-rayed.

Besides the fractures already mentioned there are two others of the lower end of the radius, Barton's fracture and fracture of the styloid of the radius. Barton's fracture (Fig. 89) seen only in the adult, is a transverse fracture of the radius just below the joint, that is, between the site of a Colles's fracture and the articulating surface of the radius. This fracture takes place at the site of what would be the epiphyseal line if it were present. There is seldom any displacement.

Barton describes this as a common fracture, but in the writers' experience in over ten thousand fractures around the wrist joint, it was found to be very rare.

Fractures of the styloid of the radius (Fig. 90) are quite common and vary in position from the tip of the styloid to an oblique fracture through the joint surface, involving a third of the radial end. If the fracture is simple it is seldom displaced. It is frequently associated with a Colles's fracture, and then it is often displaced. Ununited fractures in this region do not occur.

WRIST. Fracture of the scaphoid (Fig. 91) is the most common of all carpal fractures. It may occur alone, but is frequently associated with fractures of the lower end of the radius. It was formerly believed to be quite rare, but careful examination of *x*-ray plates has shown that it is relatively common. Fracture of this bone is infrequent in the young. Between the ages of twenty and forty it is generally unassociated with fracture of the radius. After forty it is seldom simple, but is generally associated with Colles's fracture, Fig. 92. One of the fragments may or may not be displaced. The union is

generally fibrous, so that it is impossible to determine by the *x*-ray whether it has united or not, as no callus is ever thrown out. When union does not occur, the fragments may remain in apposition for years and then some slight twist of the wrist dislocates one of the fragments. The writers have seen one case where the fragment was dislocated eight years after the original injury.

Fracture of the semilunar is next in frequency but is relatively rare, dislocation being more common.



FIG. 91.



FIG. 92.

FIG. 91.—Fracture of the scaphoid of the wrist without displacement.
FIG. 92.—Colles's fracture associated with fracture of the scaphoid; one of the fragments is displaced.

The remainder of the carpal bones are but infrequently fractured.

HAND. Fractures of the metacarpal bones are common accidents but not so frequent in the young unless there has been a crushing injury. Epiphyseal separation is most uncommon. By far the greater majority of metacarpal fractures occur after the union of the epiphysis, and they are much more common in the male than in the female. The second, fourth

and fifth metacarpals are more frequently broken. The third is seldom fractured. The fractures are of two varieties; the most common is just below the head of the bone with the head displaced toward the palmar side of the hand, Fig. 93.

The oblique fracture (Fig. 94) generally involves the shaft and may extend almost the entire length of the bone; there is seldom much, if any, displacement.

The shaft of the first metacarpal is seldom broken. When a fracture occurs in this bone it is a short oblique one just







FIG. 93. FIG. 94. FIG. 95.

FIG. 93.—Fracture of the distal head of the first metacarpal bone, with displacement.

FIG. 94.—Oblique fracture of a metacarpal bone without displacement.

FIG. 95.—Fracture of the base of the first metacarpal with angulation.

above the base, starting at the inner side and extending downward to the outer side of the bone, Fig. 95. This triangular fragment remains *in situ*, but the shaft is displaced outward, simulating a dislocation. It is difficult to reduce. When union takes place, function is not impaired, but there may be deformity.

Fractures of the phalanges (Fig. 96) are generally the result of direct violence and are frequently multiple; some, therefore, may be unrecognized. The fragments are often displaced. These fractures are relatively rare in the young, but are most common in men between the ages of twenty and forty, as that period may be termed the period of hazardous employment. These fractures always unite, but, since they are due to crushing injuries, they are either compound or are attended



FIG. 96.



FIG. 97.



FIG. 98.

FIG. 96.—Longitudinal fracture of the terminal phalanx of the thumb.
FIG. 97.—Crushing fracture of the terminal phalanx with osteomyelitis.
FIG. 98.—A small fragment of bone broken off from the articulating surface of the terminal phalanx, producing the "base-ball finger."

with lacerations, so that osteomyelitis is a common sequela, with the joint eventually becoming involved, in which event an ankylosis (Fig. 97) may ensue. The first and second phalanges are the ones most commonly broken.

Besides these fractures from crushing injuries there is also the "baseball finger," Fig. 98. When such a finger is examined by means of the *x*-ray a small fragment of bone is usually found broken from the articulating surface of one

of the phalangeal joints. The fragment is generally pulled away and union does not take place. It frequently acts as a foreign body, and a fibrous ankylosis of the joint may take place.

Fractures of the Trunk

RIBS. Fracture of the ribs is probably the most frequent of all injuries. It may occur at any age, but is most common



FIG. 99.—Fracture of several ribs with slight displacement.

in the second and third age periods. Such fractures are generally the result of falls, and while any rib may be involved it is very unusual to see the first rib broken. From the third to the ninth rib is the seat of the usual fracture; above and below that area fractures are less frequent. They generally



FIG. 99*a*.—Fracture of the sternum showing lateral displacement

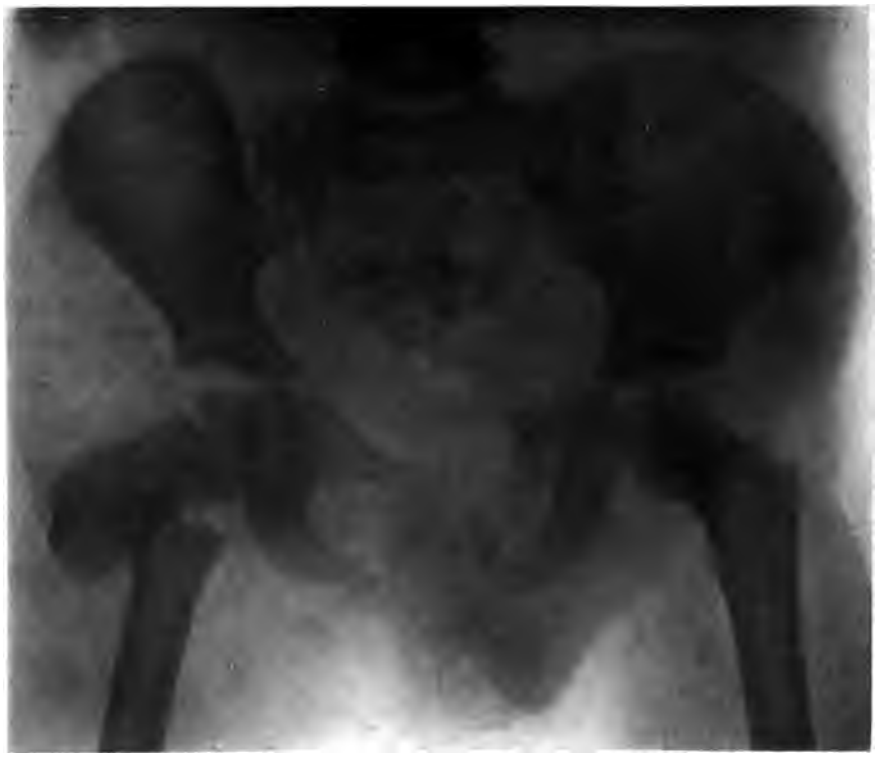


FIG. 100.—Fracture of the upper third of the femur, ilium, ischium and pubis, due to the passage of a heavy wagon wheel over the pelvis.

occur on the anterior and axillary side of the chest, though occasionally the fracture may be posterior, close to the spinal articulation. They are generally subperiosteal in character when one rib is broken, Fig. 99. If several ribs are broken they may be displaced and one of the fragments may even tear or puncture the pleura. In one case under the writers' observation resulting from a fall from a horse, the third, fourth and fifth ribs were broken at about one and one-half inches from the spinal articulation. The short fragments pierced the muscles and could be felt lying under the skin.

Fractures of the costal cartilage are quite common, but unless ossification has taken place this condition cannot be

demonstrated by means of the *x*-ray. Fracture of the ribs may occur and the position be such that it cannot be demonstrated by the *x*-ray. When no fracture can be demonstrated it is not wise to make a positive diagnosis that a fracture is not present. In such cases it has always been the writers' habit



FIG. 101.—Fracture of ischium and pubis. With this type of fracture there is seldom displacement.

to return a diagnosis of "no fracture can be demonstrated."

STERNUM. Fractures of the sternum are relatively rare and very difficult to demonstrate, as it is hard to get a clear view of the sternum. The manubrium is most commonly broken and the fracture is of the depressed variety. Lateral displacement rarely if ever occurs, Fig. 99*a*.

PELVIS. In the first age period fractures of the pelvis from simple injuries are most uncommon. In severe trauma, such as crushing injuries or passage of heavy vehicles over

the body, while almost any type of fracture may occur, separation along the epiphyseal lines is most frequent, Fig. 100.

It is in the second age period that we find the greatest percentage of fractures. Fracture of the pubis is probably the most common and generally takes place in its mid portion



FIG. 102.—Fracture of the ilium by a crushing injury.

with but little displacement. The fracture of the ischium is next in frequency. The fracture is generally near the pubis and may be accompanied by displacement, Fig. 101. Fractures of the iliac bone are next in frequency, occurring usually along the long axis of the bone, Fig. 102. In severe crushing accidents the line of fracture is through the acetabulum and the lower half of the acetabulum is displaced. In one instance in a

fall from a height where the patient landed on his feet, the head of the femur was driven completely through the acetabulum, Fig. 103.

In the third age period fractures of the pelvis are not so common and when they do occur the pubis and ischium are most frequently involved. Since most of these fractures occur



FIG. 103.—Fracture of the acetabulum where the head of the femur has been driven through and is resting in the pelvic canal.

in the hazardous occupations it follows of necessity that the vast majority of such injuries are sustained by males. There is one type of injury, however, that is quite common in the female, especially in the second age period and that is separation of the symphysis due to child birth. These separations may vary from one quarter of an inch up to one inch.

CHAPTER V

FRACTURES OF THE LOWER EXTREMITIES

CHAPTER V

Fractures of the Lower Extremities

FEMUR. *Upper End.* In dealing with injuries of this portion of the femur, the age is such an important factor that attention is again called to the three age periods:

1. Up to the union of the epiphyses.
2. From union of the epiphyses up to forty years of age.
3. Beyond the age of forty.

In considering injuries of the first age period, as has been so frequently pointed out, we have the ununited epiphysis, and this epiphyseal line constitutes the weakest portion of the bone at that point. It is a well-known dictum that a chain is only as strong as its weakest link, and the weak link in the neck of the femur is its epiphyseal line. Consequently, when an injury occurs in this region, an epiphyseal separation is expected and not a fracture of the neck, and in the experience of the writers this is what happens. The epiphyseal head is torn off and remains in the acetabulum, Fig. 104. It has rotated slightly and the shaft has ridden up. The separation is seldom complete, so that if untreated union will always take place; but on account of the rotation of the head and the riding up of the shaft there will be a shortened leg and limitation of abduction. The capsule and neck are much stronger than the epiphyseal line, so that dislocation or fracture of the neck is extremely rare.

While various authorities state that the neck of the femur in the young is fractured more frequently than is commonly supposed, yet in the writers' experience it is most uncommon. Only one such case has come under our observation. We are excluding crushing injuries, such as those caused by the weight a heavy vehicle or by falls from heights.

In the second age period fracture of the neck is also uncom-



FIG. 104.—The epiphyseal separation of the head of the femur.



FIG. 105.—Fracture of the neck of the femur just behind the head.

mon, though fracture of the head has been observed. The capsule in this period seems to be the weakest link and dislocation is seen oftener than fracture.

It is in the last age period that most of the fractures of the neck occur, and they are more common in the female than in the male. In one winter month the writers saw fifteen such fractures, of which thirteen were in the female. This percentage is abnormally high, but indicates the larger proportion in the female. The neck may be fractured where it joins the head (Fig. 105), in its mid portion (Fig. 106), or at its base, Fig. 107. The fragments may be impacted or lying free; when free the *x*-ray will show the riding up of the shaft and the trochanter



FIG. 106.—Old fracture of the mid portion of the neck of the femur with absorption.

rotated posteriorly indicating the outward rotation of the leg. Non-union is a frequent occurrence, and the frequency of non-union increases as the location of the fracture nears the head. When the fracture remains ununited there is a fairly rapid absorption of the neck. At the end of six months such fractures have been observed where the neck has been completely absorbed, Fig. 108. When the fracture is just behind the head or in the middle of the neck, impaction is relatively rare. Fracture at the base is generally impacted, and there may be more or less coxa vara. Impacted fractures without coxa vara are sometimes extremely difficult to recognize. If a second *x*-ray



FIG. 107.—Fracture through the trochanter, involving also the lesser trochanter (intertrochanteric fracture).



FIG. 108.—Old, ununited fracture of the neck of the femur with complete absorption of the neck.



FIG. 109.—Healed fracture of the neck of the femur. No callus could be demonstrated.

examination is made at the end of two weeks, the absorption at the ends of the fragments appears, and then the condition is easily recognized.

Another difficult problem for the roentgenologist to de-

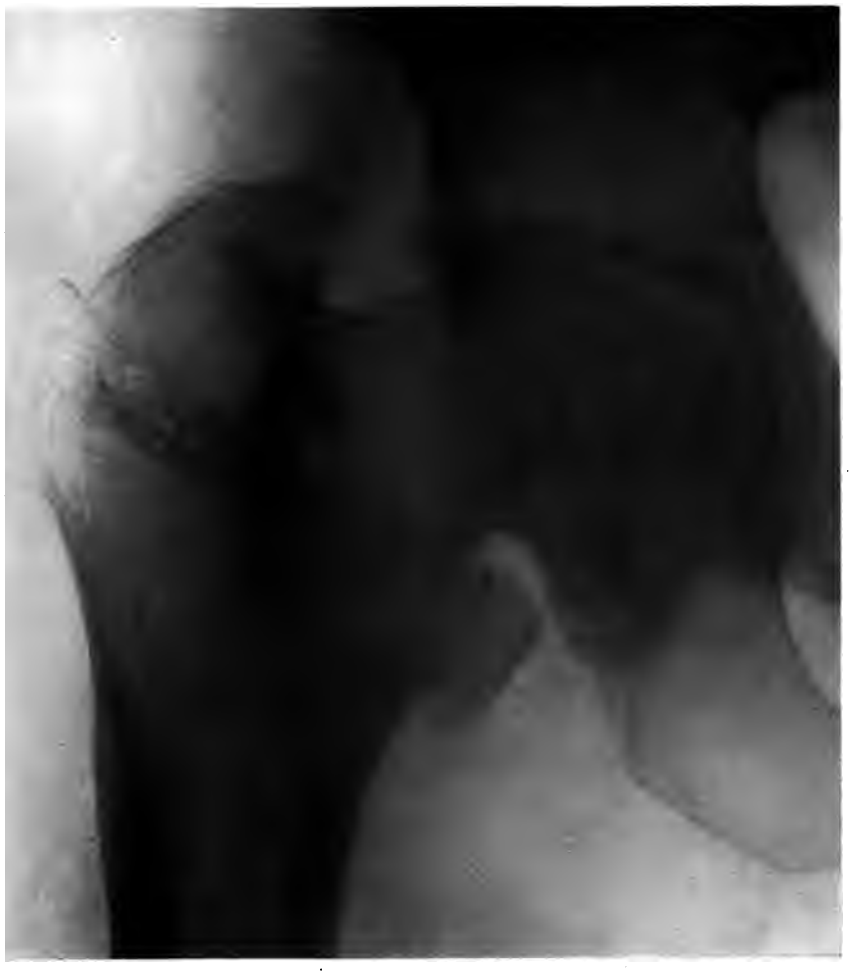


FIG. 110.—Intertrochanteric fracture with coxa vara.

termine is whether union has taken place and whether it is safe to place weight upon the leg. As the union in these fractures is sometimes fibrous it is impossible to determine that point in such cases. Even when bony union takes place there may be no

visible callus thrown down, and again the roentgenologist is placed in a quandary, Fig. 109. It has been the writers' practice never to return an affirmative opinion unless bony callus can be demonstrated.



FIG. 111.—Characteristic position of a fracture of the upper third of the femur. This was a compound fracture into which Dakin tubes had been introduced.

In considering these fractures of the neck of the femur according to the age periods the following approximate "law of probabilities" can be deduced. In the first age period, trauma generally produces an epiphyseal separation and rarely a fracture of the neck or a dislocation. In the second age period dislocation is the most common injury and fracture is uncommon. In the third age period fracture is very common and dislocation is uncommon.

Fractures involving the trochanters are generally seen in

the third age period. The fracture is generally oblique, starting through the greater trochanter or at its junction with the neck and extending downward and inward to a point just below the lesser trochanter, the lesser trochanter frequently forming a fragment by itself, Fig. 110. There is generally no displacement, but a riding up of the shaft and a resulting coxa vara. Union

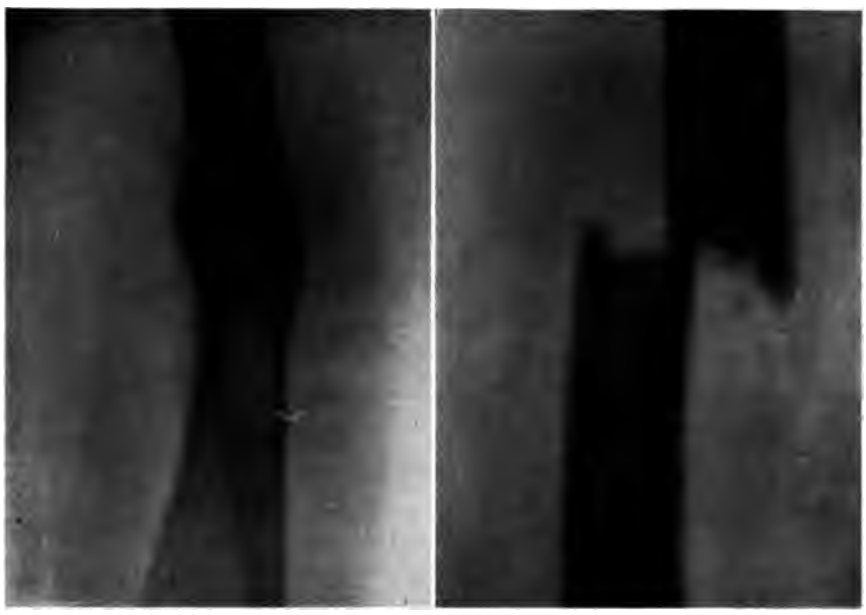


FIG. 112. FIG. 113.

FIG. 112.—Green-stick fracture of the femur which in reduction was made complete. Note the callus formation.

FIG. 113.—Transverse fracture of the femur with characteristic displacement.

always takes place and is bony in character. Fracture of the trochanters alone is unusual.

Shaft. Fracture of the upper third of the femur is quite common (Fig. 111), and the fragments assume a characteristic appearance, that is, the upper fragment is displaced anteriorly and outward while the lower fragment is posterior and inward. The separation and angulation may be so marked

that untreated fractures may result in non-union. The fractures in this region are generally oblique and may occur in any of the age periods. The only difference observed is that transverse fractures occur more frequently in the first age period.

Fractures of the middle third of the shaft are common in all age periods. They are generally oblique or green-stick (Fig. 112) in the first age period. In the second and third age periods the fracture is frequently transverse and there is generally more or less displacement, Fig. 113. Non-union is frequently due to faulty position arising from muscle and periosteum lying between the fragments.

Lower End. In the first age period the lower epiphysis is sometimes displaced. When this happens the epiphysis is generally displaced anteriorly (Fig. 114), occasionally posteriorly and the periosteum is frequently torn away from the shaft for a distance of several inches, Fig. 115. The shaft is displaced posteriorly due to the pull of the gastrocnemius. This epiphyseal separation is uncommon and seems to be associated with great violence. It is interesting to note that in the writers' series of cases three resulted from the leg being engaged between the spokes of a revolving wagon wheel.

Oblique fractures are occasionally seen occurring just behind the condyles, the fracture extending from the front backwards.

In the second and third age periods as in the first age period oblique fracture occurs just behind the condyles. If the fracture is above the attachment of the gastrocnemius the lower fragment will be posterior. Occasionally this fracture is associated with a fracture through the condyles into the joints, Figs. 116 and 117. The condyles may be separated with the shaft lying between. Occasionally there may be a fracture of one condyle alone.

In severe sprains, occasionally the ligamentous attachment will be pulled off, carrying with it a small fragment of bone.

PATELLA. Fractures of the patella are rarely seen in the first age period, and while occurring in the second age period

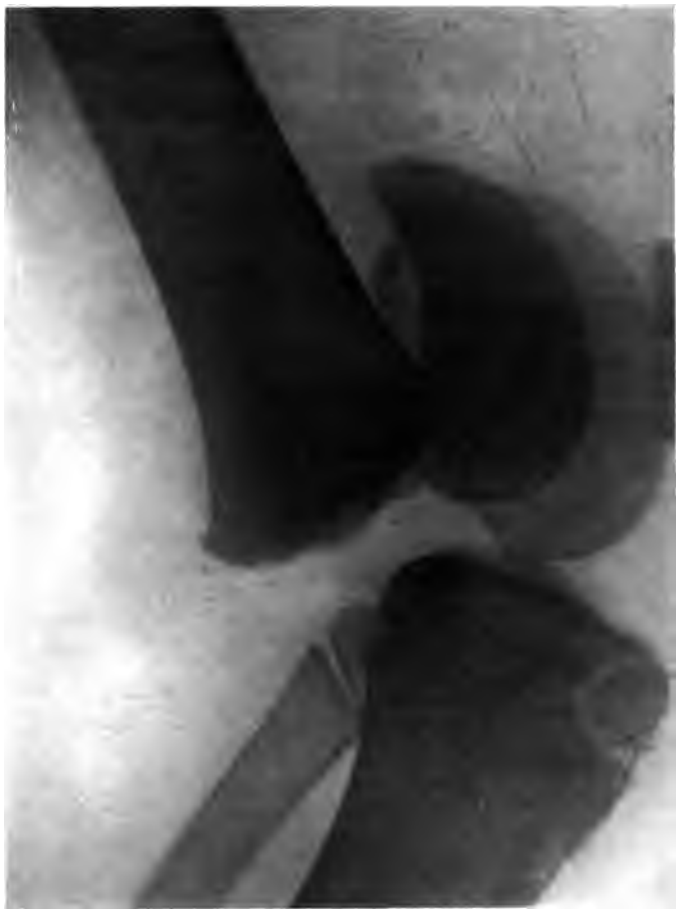


FIG. 114.—Epiphyseal separation of the lower end of the femur with the epiphysis displaced anteriorly.



FIG. 115.—Old epiphyseal separation of the lower end of the femur with the epiphysis displaced posteriorly. Note stripping up of the periosteum with new bone formation.

are most common after forty. They are much more frequent in the male than in the female. They generally result from a fall upon the knee; but the writers have seen such fractures resulting from violent contractions of the quadriceps.



FIG. 116.—Oblique fracture of the lower end of the femur associated with a fracture through the condyles and into the joint.

In those resulting from falls the fracture may be simple or comminuted, depending upon the severity of the injury. The fractures are generally anteroposterior, and the anterior third is the most frequently fractured.

If the quadriceps fascia over the patella is intact there

is seldom displacement, Fig. 118. When it is torn the fragments may be widely separated (Fig. 119), the lower fragment sometimes resting over the articulating surface of the tibia and rotated. When the fragments are approximated the



FIG. 117.—Lateral view of Fig. 116 showing the line of fracture.

union is generally fibrous and less frequently bony. There again it is difficult for the roentgenologist to determine whether proper union has taken place.

TIBIA AND FIBULA. *Upper End.* In the first age period, although the epiphyses are present, dislocation of them is most unusual. The writers have never had such a case under

observation. The tibial tubercle, however, has a separate center of ossification, and this may be pulled loose by muscular



FIG. 118.



FIG. 119.

FIG. 118.—Fracture of the patella without displacement as the fibrous sheath of the patella is intact.

FIG. 119.—Fracture of the patella with wide separation of the fragments due to a torn sheath.



FIG. 120.—Fracture of the tibial tubercle.

violence or broken by direct violence. The writers have seen several instances where the tubercle was broken by falling on a sharp edge, as the edge of a curb stone, Fig. 120.

Fractures through the heads of the tibia and fibula are unusual in this age period. In the second and third age periods fracture of the heads of these bones is quite common, and with the tibia there is generally a fracture of one of the tuberosities, Fig. 121. The tuberosity is, as a rule, forced outwards and frequently is displaced slightly posteriorly. Sometimes we have the tuberosity broken off without a transverse fracture



FIG. 121.—Fracture of the tuberosity of tibia.

of the tibia. Fractures of the head of the fibula may occur without a fracture of the tibia, but they are more frequently associated with fracture of the middle third of the shaft of the tibia, Fig. 122.

In injuries around the knee joint the tibial *spines* should always be carefully inspected, as one or both may be broken. This injury is much more frequent than has been suspected (Fig. 123), and a routine *x*-ray examination should always

be made in slight injuries or sprains of the knee. This fracture is frequently associated with dislocations of the semilunar cartilages.

Shaft. Fractures of the shaft are common in both bones, and are generally associated. Fracture of the tibia alone is quite common, while fracture of the shaft of the fibula alone



FIG. 122.—Fracture of the upper end of the fibula associated with fracture of the middle third of the tibia. (Mottled effect due to an unusually dense plaster cast.)

is rare. When the fibula is fractured alone the injury is either at the upper or lower end. There is no one definite fracture of the shaft of the tibia. It assumes a variety of forms. In the first age period it may be of the green-stick variety or oblique, Fig. 124. At the end of the first age period and the beginning of the second age period the fractures are oblique (Fig. 125) or spiral (Fig. 126) and occasionally transverse (Fig. 127),

while in the third age period they are generally transverse and frequently comminuted. In the first two age periods non-union is uncommon, while in the third age period non-union occurs quite frequently.

Lower End. In the first age period separation of the epiphyses of the tibia with or without displacement is



FIG. 123.—Fracture of the external tibial spine, frequently mistaken for a simple sprain.

quite common, Fig. 128. Separation of the epiphyses of the fibula is quite uncommon. When the epiphyses of the tibia is dislocated it is generally associated with a fracture of the lower end of the fibula. In this period linear fractures of the tibia are also seen extending from the joint upwards into the shaft, but with no displacement. In the second and third age periods fractures around the ankle joint are extremely common. The lower three inches of the fibula (Fig. 129) is the site of more fractures than any other portion of the bones of the leg. The fracture is generally oblique, and may or may not be displaced. When displaced the lower fragment is usually posterior.



FIG. 124.



FIG. 125.

FIG. 124.—Green-stick fracture of the tibia in the first age period.
FIG. 125.—Oblique fracture of the tibia in the first age period.



FIG. 126.



FIG. 127.

FIG. 126.—Spiral fracture of the tibia in the first age period.
FIG. 127.—Comminuted fracture of tibia and fibula in the third age period.

When both bones are broken Pott's fracture is the most common, Fig. 130. This is a fracture of one or both malleoli of the tibia and of the lower end of the fibula. With this fracture there is extensive tearing of the ligaments and there will often



FIG. 128. FIG. 129.

FIG. 128.—Slight epiphyseal separation of the lower end of the tibia with a fragment of bone torn from the tibia.

FIG. 129.—Fracture of the lower end of the tibia.

be a dislocation of the foot, Fig. 131. These fractures are generally associated with more or less deformity.

In examinations of the ankle joint special attention should be given to the shadows of the soft tissues in the lateral view, as they will frequently aid in determining the presence or absence of injury or disease. In the lateral view a black triangle

(Fig. 132) is formed by the os calcis, the tendo Achillis and the posterior tibial muscles. The os calcis forms the base of the triangle, and the apex is about three inches above where the belly of the muscle begins. This triangle is normally filled with fat, and offers very little resistance to the *x*-ray, so that it appears black upon the plate. Any injury or disease will



FIG. 130. FIG. 131.

FIG. 130.—Typical Pott's fracture, anteroposterior view.

FIG. 131.—Lateral view of a Pott's fracture associated with a posterior dislocation of the foot.

cause it to become filled with blood or inflammatory tissue, and the triangle becomes obliterated, Fig. 133. When the triangle is absent the plates should be carefully examined, as this is an indication of injury or disease.

BONES OF THE FOOT—ASTRAGALUS. Fracture of the astragalus is generally the result of a fall from a height, followed by a landing on the feet, Fig. 134. The fracture is generally transverse, and displacement depends upon the degree of violence. It occurs usually in the second and third age periods.



FIG. 132.—This print shows the clear triangle formed by the tendo Achillis and the posterior tibial muscles in a normal ankle joint.



FIG. 133.—Triangle obscured by swelling due either to injury or to disease.

Following injuries of the ankle joint one frequently sees a small fragment of bone lying close to the posterior border of the

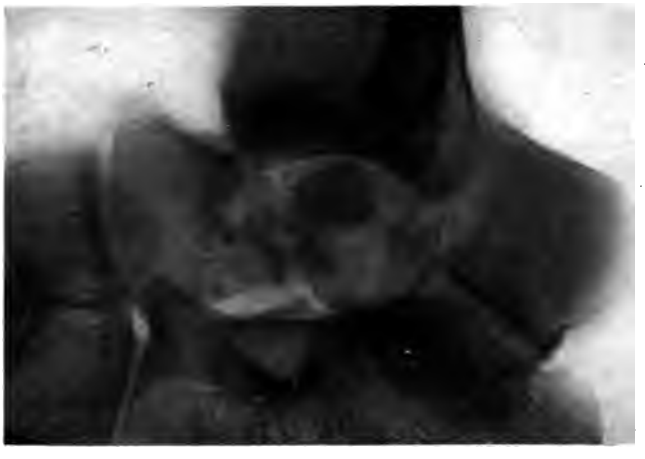


FIG. 134.—Fracture of the astragalus with separation of the fragments



FIG. 135.—Fracture of the end of the astragalus simulating an os trigonum.

astragalus. Many writers claim this fragment to be a small independent bone, namely the os trigonum. In a number of cases in our series the writers were fortunate enough to have by chance plates of the ankle before injury showing no os trigonum,

and a later plate following an injury showing this fragment, indicating that this was the result of the injury, Fig. 135.

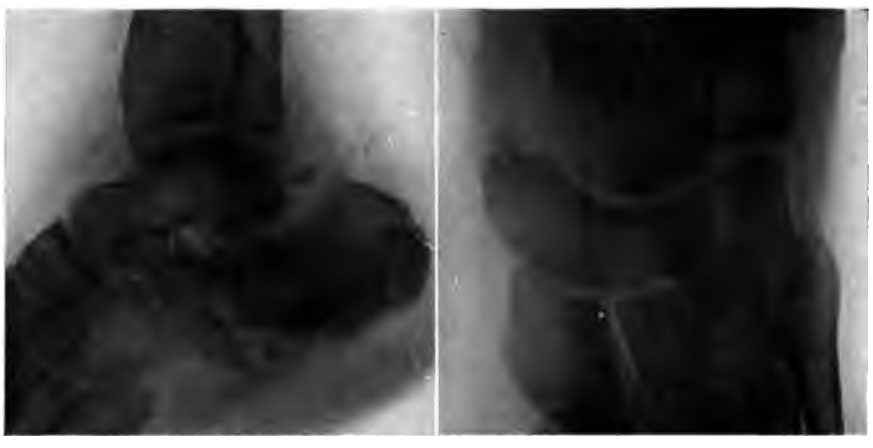


FIG. 136. FIG. 137.

FIG. 136.—Fracture of the os calcis due to a crushing injury.
FIG. 137.—Fracture of the tip of the scaphoid. Most frequently seen in toe dancers.

OS CALCIS. Like the astragalus this bone is broken in falls from heights, Fig. 136. In the writers' series of cases it was found that the majority of such fractures were sustained by carpenters, bricklayers, riveters, etc., whose occupations necessitating work upon scaffolds. This also indicates that these fractures occur in the second and third age periods. These fractures may be simple or comminuted according to the severity of the trauma. In several instances violent muscular tension has caused the tendo Achillis to pull off a fragment of bone at the attachment.

SCAPHOID. Fracture of the scaphoid is seen frequently in crushing injuries. Two cases have been observed where the internal portion was pulled off in dancers performing the ballet, Fig. 137.

CUBOID AND CUNEIFORMS. The only fractures observed in these bones were due to crushing injuries, caused by great



FIG. 138.—Old fracture of the cuboid, with some callus formation.



FIG. 139.—Fractures of all the metatarsals and some of the phalanges due to a crushing injury.

weights falling on the foot, or the passage of a heavy wagon wheel, Fig. 138.

PHALANGES. These fractures are due to direct violence and like those in the hand may be compound and associated with infection, Fig. 139.

METATARSAL BONES. The metatarsal bones are frequent fractured as the result of falls and crushing injuries, Fig. 139. The base of the fifth metatarsal is so often fractured as to warrant especial mention. It is generally due to muscular violence, Fig. 140.



FIG. 140.—Fracture of the base of the fifth metatarsal.

SESAMOIDS. Fractures of one of the sesamoids is a rare injury. The other foot should always be examined, as sometimes three sesamoids are present and one of these might be mistaken for a fragment in the injured foot.

NON-UNION

In the beginning of the previous chapter we outlined in a general way the conditions that lead up to the production of fractures, and how age, sex and occupation play an important

rôle. There is one other condition to which attention must be called and that is non-union. This is frequently encountered, and certain bones are more subject to it than others.



FIG. 141.—Old fracture of tibia with bone graft showing atrophy and smooth edges in an ununited fracture. Fragments of fibula removed. (Taken through a heavy plaster cast.)

How can an old ununited fracture be differentiated from a fresh one where sufficient time has not elapsed for callus to form? In an old ununited fracture extreme atrophy of bone and soft tissues are found and the broken ends are smooth, having lost the irregular serrated edge of the fresh fracture, Fig. 141. In a recent fracture there is but little atrophy of the bone and soft tissues. By the time atrophy begins in a fresh fracture callus should be visible, and a mistake in diagnosis should not be made.

Changes are seen upon a plate that will aid in the recognition of some of the causes of non-union. The most obvious one is where the fragments are so widely separated that union cannot take place. In other fractures, there will be actual necrosis of the ends of the bone, due to the rupture of a blood vessel, thus bringing about non-union. In certain lesions, such as sarcoma and carcinoma, the destruction of the bone will produce a fracture, and it will of necessity not unite. In the virulent stage of an acute osteomyelitis a fracture, if present,

will not unite until the lesion is halted. When the bones are in good alignment but separated by a space, shown upon the plate, that generally means that muscle or fascia is in between the fragments, and again there will be no union. There are, however, a certain percentage of cases where none of the above causes exist, but in which union will not take place. We do not know the cause, but it must be due to some alteration in metabolism probably secondary to faulty blood supply. While non-union may occur at any age if the fragments are widely separated, it is in the second and third age periods that it is most frequently met with. Probably the changes due to old age are an added factor to other unknown causes.

The humerus and femur are the two bones in which this condition occurs most frequently, and the ununited fractures of the neck of the femur are the most common. Probably one of the causes in fracture of the neck of the femur is the non-approximation of the fragments; but even when operated upon and the fragments pegged, a large percentage of cases fail to unite. The blood supply in this area is not particularly good, and this may be one of the factors.

Bone Splints. Since the roentgenologist has to deal with non-union, the operative procedures to relieve this condition must of necessity be examined also, so he must be thoroughly familiar with the various bone splints. Special attention is called to the intermedullary bone graft. This graft, generally taken from the tibia, is introduced into the medullary canal, and at first is a tight fitting bone splint (Fig. 142); but as time goes on a vacuolated area from pressure atrophy forms around the splint, especially at each end. The writers have seen this mistaken for an osteomyelitis when in reality it is merely a pressure absorption, Fig. 143.

Do not look for union of the intermedullary graft and bone, as this does not take place; the graft only acts as a mechanical splint, and is eventually absorbed. The graft at times may be broken while *in situ* and this should always be looked for and reported if present, Fig. 144.



FIG. 142.—Bone transplant of the upper end of the humerus. The transplant employed is the upper end of the fibula.



FIG. 143.—Pressure atrophy around the ends of the bone graft. (Taken through a heavy plaster cast.)

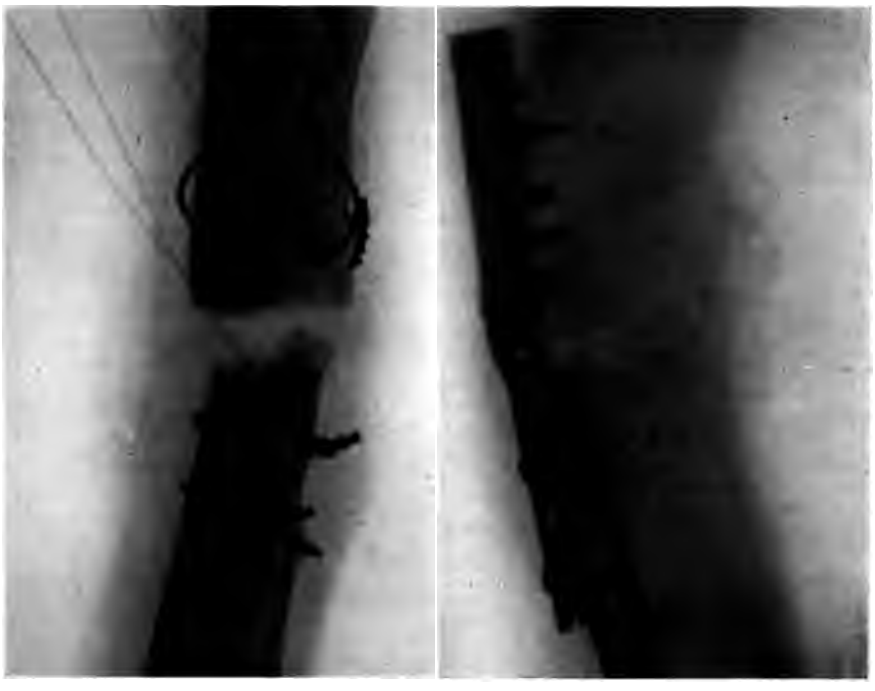


FIG. 144. FIG. 145.

FIG. 144.—Old fracture of the humerus with bone graft which is broken and partially absorbed.

FIG. 145.—Metal plate which has worked loose and is now acting as an irritant.

Other splints, such as Lane plates, wires, etc., should be carefully examined to see whether the plate is firmly attached. The splint may work loose and act as an irritant, Fig. 145.

The roentgenologist should also be familiar with the appearance of the tibia after a piece of bone has been removed for grafting purposes, Fig. 146. The area from which the bone has been removed may be mistaken for some pathological condition.

Where there has been surgical interference in a fracture it has been the experience of the writers that repair takes place more slowly and consequently callus does not appear

as early as one would expect. In cases of non-union after surgical interference, if union takes place repair is a very slow process. The writers have seen a year elapse before any



FIG. 146.—Tibia after a bone graft has been removed.

callus could be demonstrated. The roentgenologist should be very guarded in his expression as to whether union will eventually take place or not.

CHAPTER VI

CONGENITAL DISLOCATIONS

CHAPTER VI

Congenital Dislocations

WHILE congenital dislocations have been noted in many of the joints, yet with the exception of the hip they are uncommon. The etiological factor has not been definitely established, but some malposition of the parts in fetal life may be the causal factor. In the newborn the heads of the bone are entirely cartilaginous and cannot be demonstrated by the *x*-ray, so in some cases injuries at birth may deform or displace the head, and later on these dislocations be termed congenital. Congenital dislocations of the hip cannot, however, be attributed entirely to birth injuries, as under such conditions the percentage of cases should be equally distributed between the male and female, whereas the lesion occurs much more frequently in the female.

Congenital Dislocation of the Hip. This condition is seldom recognized at birth. The abnormal condition of the hip is only noted some time after the child has started to walk. The waddling gait at first is thought to be simply awkward efforts in learning to walk. When this does not clear up medical advice is sought. The fact that the child is seen after it has learned to walk has an important bearing upon the *x*-ray findings. In studying the plate of such a case, there are several points which should be carefully noted—the shape of the pelvis, the position and shape of the head and neck of the femur, and the size of the affected bone in comparison with the normal one.

In a newly born child and until walking takes place, the pelvis, roughly speaking, is triangular in shape, with the base above and the apex at the pubis. The two sides of the triangle are approximately straight (Fig. 147); but when the child walks, the upward thrust of the femur localized at the ace-

tabulum, which is the point of articulation, gradually causes the acetabulum to be pushed in slightly, and at this age, the bones being soft, this is easily accomplished. At the same time the body weight exerting a downward pressure causes the ilia to flare out. These two mechanical factors change the shape of the pelvis, so that where it was triangular with straight sides before walking, it has now become a triangle with the base slightly widened and the sides slightly curved, the convexity being inward, Fig. 148. This naturally gives a stronger joint and one in which dislocation is less apt to occur. When one hip is dis-

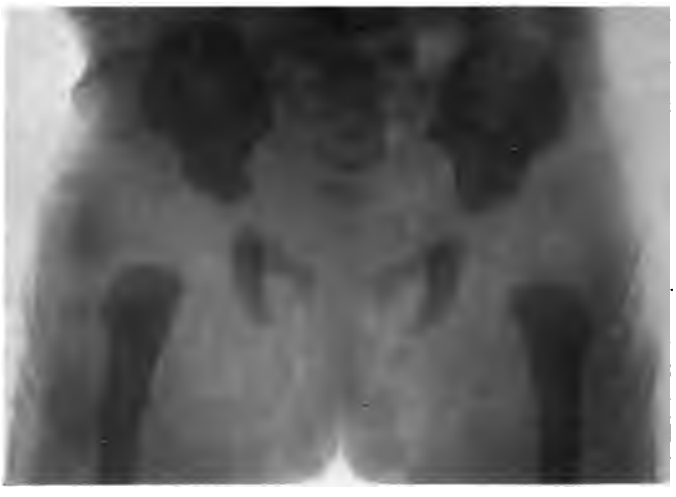


FIG. 147.—Straight sides of a pelvis in a child who has not walked.

located that side of the triangle will be found straight and the other slightly bent in. The acetabulum will also be shallow, as there will have been no pressure from the femur to deepen it, Fig. 149. The dislocation is generally upward, so the trochanter will be high. The neck is also frequently bent, and this may throw the head anteriorly and, from its appearance upon the plate, suggest that it may have been destroyed by disease. Congenital dislocations are found at the age when acute epiphysitis, tuberculous and non-tuberculous, is prevalent. In these two conditions, however, not only will the head be partially or wholly destroyed, but the acetabulum will also



FIG. 148.—Beginning concavity of the sides of the pelvis due to the upward thrust of the femora in walking.



FIG. 149.—Congenital dislocation of the hip. The acetabulum is shallow and there is no concavity of the pelvis on the affected side. Note the distance of the dislocated head of the femur from the acetabulum.

be more or less involved, Fig. 150. In congenital dislocation the acetabulum will be shallow and the outlines clean-cut, but there will be no destruction. If the disease takes place after the child has walked there will also be the bent side of the tri-



FIG. 150.—Destruction of the head of the femur with pathological dislocation. The acetabulum is also involved. The destruction allows the neck and shaft to lie close to the pelvic wall with no intervening space. Compare this with the space seen in congenital dislocation, Fig. 149.

angle, while in dislocation it will be straight. Then, too, in the non-tuberculous epiphysitis there will be new bone formation in the late stages and this is never present in dislocation, Fig. 151. Occasionally one will find a new small acetabulum on

the posterior surface of the ilium in dislocation. In diseased processes, as a rule, the trochanter, besides being high, is closer to the bone on account of destruction, while in dislocation a wide space generally separates the femur from the



FIG. 151.—Bony ankylosis seen in non-tuberculous infections.

pelvis. In congenital dislocation the femur on the affected side will be smaller than the normal one, due to retarded development probably from lack of bone stimulation. This, however, is also seen when the head has been destroyed by disease, Fig. 152.

Congenital dislocation of the hip is much more common in the female, being in the proportion of six or seven to one in the male. Either hip may be involved; in the writers' series of cases the distribution was about equal. Dislocation of both hips



FIG. 152.—The small undeveloped femur in congenital dislocation. This is not "atrophy of quantity" but non-development.

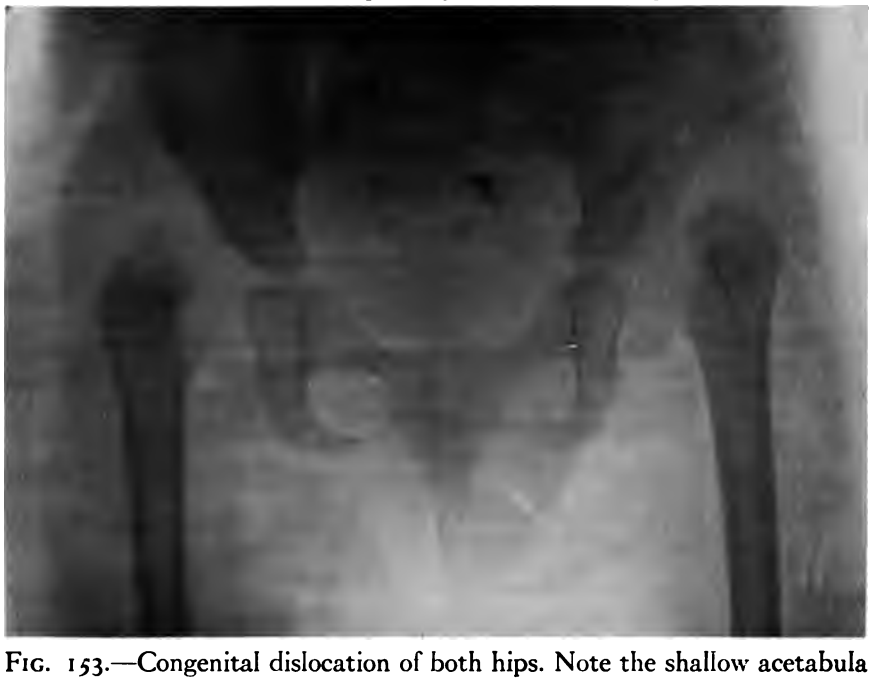


FIG. 153.—Congenital dislocation of both hips. Note the shallow acetabula and straight sides of the pelvis.



FIG. 154.—Injury of the lower epiphysis with consequent retardation of growth causing a partial subluxation of the ankle joint.



FIG. 155.—Club foot.



FIG. 156.—Club hands.



FIG. 157.—Abnormal position of femur due to anterior poliomyelitis. This hip could be dislocated with but slight manipulation.

occurred in about 30 per cent of the total number of cases, Fig. 153.

Dislocations of the Shoulder Joint. In dislocations of other joints birth injuries play a most important part, and this is particularly true of the shoulder joint. As has already been stated, injuries to the humeral head may occur without being recognized and only be discovered later in life. Obstetrical paralysis may cause subluxations and these later may be called congenital.

Abnormalities in the Development of Epiphyses. Any abnormality in the development of the epiphysis, either over-development or retardation, may bring about mechanical dislocation. The writers have seen several cases where the epiphyseal head of one of the bones of the leg or forearm failed to develop. This, of course, retarded the growth of the bone, and with the normal increase in the length of the adjacent bone mechanically brought about a dislocation, Fig. 154. In club foot (Fig. 155) and club hand (Fig. 156) partial or complete dislocations may occur, just as in obstetrical paralysis we may find a dislocation of the hip due to anterior poliomyelitis, Fig. 157.

CHAPTER VII

ACQUIRED DISLOCATIONS

CHAPTER VII

Acquired Dislocations

DISLOCATIONS are not often seen by the roentgenologist before being reduced. The majority are reduced and then sent for *x*-ray examination to see if the reduction is proper or to rule out a possible fracture. In the writers' series of cases the percentage of shoulder dislocations is very small, due to the above fact. In hospital and out-patient departments, practically every fracture is seen before and after reduction, while dislocations are generally seen only after reduction.

Shoulder. In the first age period dislocations of the shoulder are rare, though occasionally one may result from instrumental delivery. The second age period shows the greatest number of dislocations, and they are largely confined to the male. This period covers the age of hazardous occupations, and since the female does not engage in such pursuits dislocations are relatively rare. The various text books of surgery give percentages covering the different types of dislocations. From personal observation the subcoracoid (Fig. 158) has been found to be by far the most common, and the variations of the subglenoid type (Figs. 159 and 160) are probably next in frequency.

Dislocations in the third age period are generally the result of falls and occur with the same relative frequency in both sexes. In this age period, however, we find that these dislocations are often associated with fractures of the greater tuberosity or of the surgical neck. It is common to see a fracture of the surgical neck with the end of the shaft in the glenoid fossa and the head dislocated outward, Fig. 161. In these cases one wonders whether the dislocation is the result of the fracture or vice versa. Occasionally one will see the head split longitudinally with a portion of it in the axilla.

ELBOW. Dislocations of this joint appear with the same relative frequency as to age and sex as those of the shoulder. They are much more common in the male, and occur most often between the ages of twenty and forty.

In the first age period dislocations of the elbow are relatively rare. In some of the older works it is stated that the great majority of the dislocations of the elbow occur under twenty and that half of them occur under the age of ten. The writers have



FIG. 158.—Subcoracoid dislocation of the humerus.

seen many cases in young children diagnosed as dislocations, in which the *x*-ray examination has shown that the condition was one of epiphyseal separation or a supracondyloid fracture. It is reasonable to suppose that the epiphyseal line is weaker than the capsule, and consequently we have separation instead of dislocation. Most of these dislocations are encountered in the latter portion of the first age period and the first half of the second age period. Dislocation of both bones backward is by far the most common, with its variations of outward and

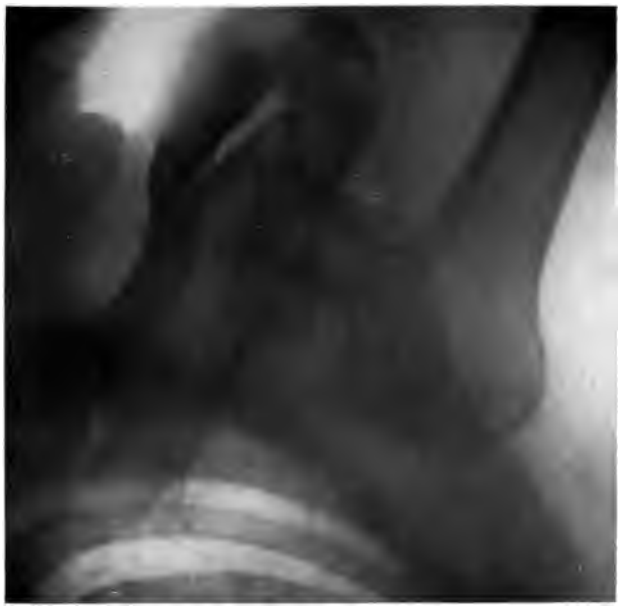


FIG. 159.—Subglenoid dislocation with unusual position of the humerus. (*Courtesy of Dr. Henry J. Walton.*)



FIG. 160.—Subglenoid dislocation. The usual position of the humerus. (*Courtesy of Dr. Henry J. Walton.*)

inward, Fig. 162 and 163. Dislocation forward is apparently a very rare condition. These dislocations are frequently associated with fractures of the coronoid, olecranon and head of the radius.

ULNA AND RADIUS. The ulna is sometimes dislocated backwards with an accompanying fracture of the radius. The radius may be dislocated forward (Fig. 164 and 165)



FIG. 161.—Subglenoid dislocation of the humerus associated with fracture, the shaft resting in the glenoid fossa. (*Courtesy of Dr. John Evans.*)

without fracture, but when dislocated forward and upward it is invariably associated with a fracture of the upper third of the ulna.

In the extensive tearing of the ligamentous attachments in dislocations there sometimes follows a partial or complete ossification of the capsule with a resulting stiff joint, Fig. 166.

In the third age period dislocations are uncommon. Injuries





FIG. 162. FIG. 163.

FIG. 162.—Dislocation of both bones of the forearm backward, associated with fracture. (*Courtesy of Dr. Henry J. Walton.*)

FIG. 163.—Another view of the condition indicated in Fig. 162, showing the lateral displacement. (*Courtesy of Dr. Henry J. Walton.*)





FIG. 164. FIG. 165.

FIG. 164.—Dislocation of the elbow associated with fracture of the neck of the radius. (*Courtesy of Dr. Henry J. Walton.*)

FIG. 165.—Same as Fig. 164, showing lateral view. (*Courtesy of Dr. Henry J. Walton.*)



FIG. 166.—Dislocation of the elbow reduced, followed by deposition of bone in the torn ligaments producing ankylosis.





FIG. 167. FIG. 168.

FIG. 167.—Dislocation of the wrist joint.

FIG. 168.—Anterior dislocation with rotation of the semilunar.

producing dislocations in the second age period produce fractures in the third age period.

WRIST. Just as in other joints, dislocations of the wrist are rare in the first age period. Since the advent of the *x*-ray many so-called dislocations are found to be fractures, and the one most often mistaken in this age period is the epiphyseal separation of the radius. The comparatively few dislocations that do occur are in the second age period, Fig. 167. Dislocation of the ulna backward is sometimes seen.

Dislocations of the carpal bones at the radial articulation or at midcarpal articulation are seen very rarely, and when they do occur are generally associated with fracture. While any carpal bone may be dislocated, the semilunar is the one by far most frequently involved. It is dislocated anteriorly and is generally rotated, so that the radial articulation is pointing backward, Fig. 168.

HAND. Dislocation of the thumb metacarpal at the carpal articulation is by far the most common of the metacarpal dislocations, and is generally backward. While the other metacarpals may be dislocated, fractures occur much more frequently. Dislocations of the phalanges (Fig. 169) are quite common, and backward dislocations of the first phalanges, especially of the thumb, are the most frequent of all.



FIG. 169.—Backward dislocation of the thumb phalanx.

PELVIC BONES. Dislocations of the pelvic bones are quite rare, though the writers have seen one case where a blow upon the sacrum caused a complete forward dislocation. The so-called sacro-iliac subluxations, in the writers' opinion, do not exist. The joint is of the saw-tooth variety and before a slipping could take place these saw-tooth edges would have to



FIG. 170.—Backward dislocation of the hip.



FIG. 171.—Obturator dislocation of the hip.

be broken. These conditions must be ligamentous sprains.

HIP. Dislocations of the hip are much more common in the male than in the female. In the first age period, except in pathological conditions, dislocations are very rare. The epiphyseal line being present, injury to this joint causes a separation instead of the tearing of the capsule followed by a dislocation.

It is in the second age period that most of the hip dislocations occur as the neck of the femur is much stronger than the





FIG. 172. FIG. 173.

FIG. 172.—Lateral dislocation of the patella.

FIG. 173.—Subluxation of the tibia, due to an old tuberculous process.

capsule, and consequently trauma causes the capsule to tear, allowing the head to slip out, instead of producing a fracture of the neck. Backward dislocations are the most common (Fig. 170), and probably dislocations into the region of the obturator foramen are next in frequency, Fig. 171. Besides these two groups there are variations of each one.

In the third age period, on account of the absorption of

the lime salts with a resulting brittleness of the neck, trauma will cause a fracture of the neck instead of a dislocation, though occasionally dislocations are seen in very old people.

PATELLA. Dislocations of the patella are uncommon, though when they do occur they are either internal or external, Fig. 172.

KNEE. Dislocation of the knee is more common in the male than in the female. This is due not to any structural difference in the knee, but to the fact that the male engages in more violent pursuits and hazardous occupations. Dislocations of the knee in any case, however, are very uncommon. They seldom occur in the first age period, due to the presence of the epiphyses. The cartilaginous union of the epiphysis is weaker than the capsule of the joint; consequently in injury it yields instead of the capsule.



FIG. 174.—Posterior dislocation of the foot with fracture.

TIBIA. In the second age period the tibia may be dislocated backward, forward or laterally. The backward dislocation is probably the most common. Complete lateral dislocations are quite rare without fracture, though slight lateral subluxation is frequently seen as the result of bad tearing of the ligaments, or one condyle may be displaced laterally in a longitudinal fracture through the joint.

In old pathological processes, such as tuberculosis, the contractions of the muscles combined with the destruction of the articulating surfaces frequently bring about a posterior dislocation of the tibia, Fig. 173.

Dislocation of the semilunar cartilage is quite a common occurrence, but since cartilage does not cast a shadow upon the plate it cannot be demonstrated. This condition is often associated with fractures of the tibial spines, and when this is noted one should also be suspicious of a dislocated cartilage.

FIBULA. Dislocations of the upper end of the fibula from injuries and from muscular violence have been reported, but they are extremely uncommon. The writers have never seen such a case.



FIG. 175.—Dislocation of the first metatarsal. (*Courtesy of Dr. Henry J. Walton.*)

Pathological dislocations, such as crushing injuries with fracture of the head of the tibia, or nongrowth of one of the bones, are occasionally found.

FOOT. Dislocations of the foot are quite common (Fig. 174), but rarely occur in the first age period, as in that period the epiphysis yields instead of the joint. In the second age period this injury is most frequent. The foot may be dislocated backward or laterally. It is often associated with a Pott's fracture. Any of the tarsal bones may be dislocated, but dislocation of the astragalus is most common. Dislocation of the metatarsals is uncommon without fracture. The first is the one most commonly involved, Fig. 175. Dislocation of the phalanges of the feet does not occur as frequently as dislocation of these bones of the hands. They are generally associated with fracture, and, as they usually result from crushing injuries, they are frequently compound.

CHAPTER VIII

BONE INFECTIONS

CHAPTER VIII

Bone Infections

THIS chapter will be devoted to the discussion of those infections that primarily involve the bone, though they may simultaneously or later involve the joint. Osteomyelitis is the most important and frequent of these infections.

Osteomyelitis. In Chapter II on Normal Bone attention was called to the constituent parts of a bone: the periosteum, cortex, medullary canal, the cancellous heads and the cartilaginous articulations. Any one or all of these structures may be involved in an inflammatory process. When the periosteum is involved we have a periostitis; when the infection is confined to the cortex we have an osteitis, and when the medullary canal is attacked alone we speak of a myelitis. The combination of a diseased cortex and medullary canal is known as osteomyelitis. While the pyogenic factors may vary in bone infection, the process is the same, varying only as to the severity and duration of the infection.

Before taking up the changes that occur it is well to bear in mind just how the infection reaches the bone, as the picture varies according to the point at which the infection starts. In a general way we may speak of four portals of entry.

1. Infection, hematogenous or lymphoid in origin, carried directly to the medullary canal by means of the nutrient canal.
2. Infection lodging beneath the periosteum.
3. Infection arising within the joint.
4. Infection by direct inoculation, as in wounds and compound fractures.

In the first group, the blood or lymph supply carries the infection through the nutrient canal into the medullary canal; and since this is filled with soft marrow fat the infection



FIG. 176. FIG. 177.

FIG. 176.—Osteomyelitis showing involvement of the medullary canal and cortical bone.

FIG. 177.—Osteitis with an extensive periostitis.

may spread easily and rapidly up and down the canal, and by means of the Haversian canals will finally involve the dense compact cortical bone, Fig. 176. In this condition the changes take place within the bone, and the infection works its way toward the periphery. In the earlier stages the cortex and periosteum are not involved.

When the infection lodges beneath the periosteum we have both it and the bony cortex involved. Since the cortex is



FIG. 178.—Here is an acute infection starting in the joint and involving the femur.

quite dense the infection spreads more easily into the periosteum and along the outside of the shaft. The cortex becomes infected more slowly on account of its density, and the spread of the disease is more or less limited. Consequently the medul-



FIG. 179.



FIG. 180.

FIG. 179.—Compound fracture with osteomyelitis.
FIG. 180.—Osteomyelitis with sequestrum lying in a cavity.

lary canal is not often involved, but with the localized osteitis we may have an extensive periostitis, Fig. 177.

When the infection starts in the joint we have extensive destruction of both articulating surfaces, and finally the disease breaks through one of the cartilaginous surfaces and

destroys to a more or less degree the head of the bone where the cancellous bone is present; and again the infection extends but slowly into the medullary canal proper. Cartilage is quite resistent to infection, and it is only the more virulent organisms, such as streptococcus, etc., that produce such conditions, Fig. 178.

In compound fractures (Fig. 179) the infection is carried directly to the medullary cavity and raw, exposed bone surfaces, so that periosteum, cortex and medullary canal may be involved simultaneously.

The question naturally arises: "Does osteomyelitis give us a constant roentgenological picture?" The answer must be, "No," but this answer may be qualified by stating that the pathologic process is the same, varying only as to the intensity and duration of the infection. Hence, if we understand the fundamental principles of bone infection, while the picture may vary, yet the condition may be recognized. Only two changes may be seen upon a plate upon which to base a diagnosis—bone destruction and bone production. The variation in these two processes constitutes the basis of a diagnosis.

Attention has been called to the fact that the dense, compact bone is pierced by numerous small Haversian canals, and that these are in direct connection with the medullary canal. Now an infection that starts in the medullary canal not only extends up and down, but also enters the Haversian canals; and since these canals extend through the compact bone and have numerous intersections the roads for the transportation of infection are already prepared. Between these canals we have dense masses of calcium salts which, relatively, are but slowly absorbed by infection. Thus these canals make it possible for the infection to spread and break out in other portions of the bone, frequently leaving normal bone between the primary focus and the secondary lesion, as the infection may have failed to enter all the canals in the affected area. Often by means of the intersecting canals the infection

may completely surround an area of bone and thus devitalize it and form what is known as a sequestrum, Fig. 180.

Since the infection is at first confined to the medullary canal and within the Haversian canals, there is a short period of time when the canals are filled but no destruction has taken place. This is important to remember, because, as has already been pointed out, the only changes visible upon a plate are bone destruction and bone production; so there is a period in beginning osteomyelitis when the *x*-ray examination will show a perfectly normal bone. Frequently cases of acute osteomyelitis have been seen in the first ten days of the infection, where there were extensive swelling and inflammation of the soft tissues surrounding the bone, with intense pain and high temperature, yet the *x*-ray examination was negative, Fig. 181. Such cases at operation will show an inflamed, infected periosteum and the canals full of pus, but no destruction. Care must be taken in such cases not to mislead the surgeon by reporting no infection present.



FIG. 181.—*X*-ray of an acute osteomyelitis one week after onset. Operation showed the Haversian canals to be full of pus, but since breaking down of the bone tissue had not occurred, the *x*-ray examination was negative.

When the infection is very virulent we have extensive destruction and but little new bone production, while in the chronic type we have but little destruction and extensive bone production, Fig. 182.

In an acute osteomyelitis we get the following picture upon a plate: The infection, having lodged in the medullary cavity, takes the path of least resistance and extends along the medul-

lary canal, and we get vacuolated spaces represented by areas of lessened density. The infection now spreads to the bony cortex and travels irregularly by means of the Haversian canals, so that there are areas of bone destruction with normal bone in between, and eventually these normal areas will be cut off and become sequestra. This is an important point of differential diagnosis, because every pathological process in bone must be viewed as a possible malignancy until proven otherwise. Malignancy in long bones starts from one central point and radiates equally in all directions, absorbing the bone as the growth advances, but never appears as separated areas with normal bone in between and never produces sequestra.



FIG. 182.—Chronic osteomyelitis showing marked new bone formation but with little destruction.

The osteomyelitic infection will finally pierce the cortex in one or more places, leaving areas of normal cortex between. Here again we have another important point of differential diagnosis, as in malignancy, when the growth reaches the cortex, it destroys it completely as a whole and not in parts as osteomyelitis does.

Thus far we have been discussing the acute destructive process; but, when the cortex is pierced and drainage established, nature, with the acquired resistance of the tissues,



FIG. 183. FIG. 184.

FIG. 183.—The new bone formation defines the boundaries of infection.

FIG. 184.—The periosteal new bone gives the appearance of a shaft actually expanded, but the apparent expansion is due to deposition of bone on the outside.

now attempts to limit the destructive process. This is done by building up a new bone wall at the edge of the infection. To produce bone reaction there must be stimulation, and the point of stimulation has to be at the point where the infection stops and the normal bone begins; so we must look for our new bone production at the edges of the infection and not in the middle of it. The result of this will be that the new bone laid down will follow the edge of the infection. This gives us a varied picture, as the boundaries of the destructive process will determine where the new bone will be laid down, Fig. 183. At the same time, since the infection has reached the periosteum, we shall find extensive periosteal bone following the course of the periosteum. This deposit of periosteal bone will frequently be sufficiently great to give the appearance of expansion of the bone, Fig. 184. Close inspection, however, will reveal that the apparent expansion is in reality due to deposition of bone on the outside. This, too, is an important point, as benign growths of the bone, such as cysts and osteochondromata, invariably expand the cortex while osteomyelitis does not.

The predominating feature in acute osteomyelitis is bone destruction with but little new bone formation. The acute types are seen most frequently in the young and in early adult life.

In chronic osteomyelitis the conditions are just reversed. We have excessive bone production with but few small areas of destruction. The entire bone is thickened, frequently to such an extent that the medullary cavity seems to be obliterated. The bone is often irregular in shape and much thickened, due to extensive deposition of periosteal bone upon the cortex. In the bone there may be small areas of absorption due to focal spots of infection. When sequestra are present they are usually very small, and, on account of the surrounding dense bone, frequently cannot be demonstrated by the *x*-ray, Fig. 185. This type of osteomyelitis is seen more frequently in middle and old age. When the infection starts beneath the periosteum the lesion will be confined to the cortex. When it starts in

the joint it spreads slowly into the cancellous head, and if untreated will reach the medullary canal and then spread rapidly.

In compound fractures the infection is confined to the ends of the bone and seldom spreads, as bone drainage from the infection exists through the open wound.



FIG. 185.



FIG. 186.

FIG. 185.—Chronic osteomyelitis with a small cavity containing a sequestrum.

FIG. 186.—Vacuolated area in the head of the radius, known as Brody's abscess.

In a certain number of cases we shall find a single punched-out area, definitely circumscribed, situated in the cancellous head of a bone about a half inch from the joint. Such a localized osteomyelitis we speak of as Brody's abscess, Fig. 186. The upper ends of the tibia and humerus seem to be the favorite locations for such conditions.

From this description it will be readily seen that there is no



FIG. 187.—A most atypical tuberculosis of the shaft of the radius in a patient of forty-five years. The condition was not diagnosed until a microscopic examination was made after operation.

one definite *x*-ray picture, but that the changes will depend entirely upon the virulence of the infection, the stage of the disease and the resistance of the patient.

The six points which follow should always be carefully noted upon the plate:

1. The place where the infection starts.
2. The character of the destructive process.
3. The path of extension, that is, spreading in all directions or following the path of least resistance.
4. The character and situation of the new bone production.
5. The condition of the cortex, whether it is intact, destroyed as a whole or pierced by sinuses, expanded or unexpanded.
6. The type of periosteal reaction, whether it is parallel or perpendicular to the shaft.

Thus far we have discussed only those infections arising from pyogenic organisms.

Tuberculosis. The question of tuberculosis as an osteomyelitic infection has been much discussed, and some authorities state that it is much more frequent than is commonly supposed. In the writers' experience it was found to be extremely rare in adults, only two cases having been encountered in which diagnosis was made only by the microscopical examination following operation. In one case there was extensive destruction of the shaft of the ulna, no new bone formation, no sequestrum, and the cortex was expanded just as in a benign growth, Fig. 187.

It is true that in extensive tuberculosis of the joint, the disease penetrates the cartilage of one of the articulating surfaces and involves the head of the bone, but the infection is generally limited to the bone beneath the cartilage and does not extend down into the cancellous bone as does an ordinary osteomyelitis, Fig. 188. There is no new bone production, no sequestrum and the joint is always involved.

In children, however, it is common to see a tuberculous dactylitis which may involve one or more bones accompanied

by a marked periostitis. Occasionally one of the metacarpals or phalanges will show punched out areas in the shaft of bone similar to cysts. Lues and osteomyelitis will frequently give the same appearance upon the plate, so that often it is impossible to differentiate these three conditions by *x*-ray examination, Fig. 189.



FIG. 188.—Tuberculosis of one side of the epiphysis of the tibia. The disease does not extend down into the cancellous portion of the bone beneath the epiphysis.

Besides the tuberculous dactylitis in children we occasionally see tuberculosis of the shaft, and this is probably more frequent than has hitherto been supposed. The writers have seen five such cases in the last year. These lesions generally start at or just behind the epiphyses in the cancellous bone or medullary canal. The bone is destroyed just as in ordinary osteomyelitis, and there may be a marked periosteal bone production but seldom new bone in the shaft. The lesion is

frequently multiple. So far, from the few cases observed, it has seemed impossible to differentiate it from an acute or luetic osteomyelitis. In the tuberculous types, however, the



FIG. 189.



FIG. 190.

FIG. 189.—Tuberculosis of the first metacarpal with marked periostitis.
FIG. 190.—Tuberculous osteomyelitis in an infant with sequestrum. This cannot be differentiated from the inflammatory type of osteomyelitis.

joint is frequently involved and the lesion is near the epiphysis, Fig. 190.

Lues. Lues, while attacking practically all the structures of the body, seems to have a peculiar affinity for the bones, and the congenital and acquired forms manifest themselves in a variety of ways. The congenital type will be discussed in the chapter on "Joint Lesions of Children." The acquired form varies from a slight periostitis to an advanced osteomyelitis. Luetic periostitis is probably the most common of all lesions, and is seen most often in the last half of the second and all of the third age period. It may involve one or several bones. The periostitis is generally marked, and the calcium salts are laid down more or less parallel to the long bone and are frequently raised a little from the shaft. This type cannot be differentiated from the inflammatory type. If two or more bones, however, are involved, the lesion is generally luetic in origin, and this is especially true if it is accompanied by an osteitis

without a cavity, Fig. 191. Occasionally the calcium salts may be laid down perpendicular to and extending out from the shaft about one quarter of an inch. For want of a better name



FIG. 191. FIG. 192.

FIG. 191.—Luetic periostitis, showing involvement of both bones of the forearm.

FIG. 192.—Luetic periostitis (lace-work type) of the ulna.

we speak of this as the "lace work type" of periostitis, and when this is present it is invariably luetic in origin. Unfortunately this type is the exception rather than the rule, Fig. 192.

Luetic osteomyelitis produces the same bone changes as the inflammatory type and frequently cannot be differentiated.



FIG. 193.—Luetic osteomyelitis without swelling of the soft tissues. Few clinical signs.

There are two points, however, that assist us in making a correct diagnosis. First, the lesions are frequently multiple and there is generally more bone production. The second point is very important—the clinical picture does not coincide with that given by the *x*-ray. In lues the plate may show a very acute osteomyelitis evidenced by extensive destruction, the entire bone being involved. With such a plate one would expect an acutely inflamed part, very tender and accompanied by fever. Instead there will be but few clinical signs, only slight tenderness, no fever and practically no inflammation, Fig. 193.

A careful study of the plate will give not only a correct diagnosis, but further information to the surgeon. It shows not only the extent of the disease and the presence of sequestra, but it also determines the presence or absence of an involucrum. This is of great importance, as it is frequently the



FIG. 194.—Marked involucrum with the entire shaft becoming a sequestrum.

determining factor as to the character of the operation necessary. For example, in an acute osteomyelitis, where practically the whole bone is involved, it will be necessary to take away the entire bone to cure the patient. Now, if the plate shows no involucrum (Fig. 195) the surgeon will confine himself to opening the bone to get free drainage, and if the patient's condition remains good the radical operation will be delayed until the involucrum has formed, Fig. 194.

Typhoid. Typhoid infection takes place in the second age period, but it is a rather rare condition. The spine and ribs are most frequently involved, occasionally the long bones, Fig. 196. When the spine is involved the margin of one vertebra is attacked close to the cartilaginous disc. The destructive process may destroy the entire disc, and when repair takes place there is marked new bone production and sometimes actual ankylosis. The writers have seen one case in which the mandible was involved. The plate showed a round punched-



FIG. 195.—An acute osteomyelitis with no involucrum but very slight deposition in the periosteum.

out area, sharply circumscribed; a dense calcium deposit surrounded the infection, indicating that the lesion was a very chronic and inactive process. Occasionally we may have a periostitis of the long bones, but it cannot be distinguished on the plate from the ordinary inflammatory types.

ACTINOMYCOSIS. Actinomycosis usually affects the jaw



FIG. 196.—A typhoid periostitis with a cavity in the cortex.

and produces a chronic osteomyelitis, with no special characteristics which make a differential diagnosis possible by *x*-ray.

RAYNAUD'S DISEASE. In Raynaud's disease (Fig. 197) the terminal phalanges are involved. Bone atrophy and marked

thinning are present giving a spearlike appearance; finally the entire phalanx may be entirely absorbed. The lesion is generally confined to the terminal phalanges. Occasionally the same changes are seen in endarteritis obliterans. In dry gangrene there is a gradual absorption of the phalanges.

LEPROSY. Leprosy (Figs. 198, 199) is characterized by a



FIG. 197.—Raynaud's disease, showing the spearlike appearance of the terminal phalanges.

progressive atrophy starting in the terminal phalanges and gradually extending backward, involving all the phalanges. There is a gradual absorption of the bone so that it eventually completely disappears.

COCCIDOIDAL GRANULOMA. Coccidoidal granuloma (Figs. 200, 201, 202), is due to a parasitic organism involving the bones, joints and soft tissues. The *x*-ray appearance of a joint is essentially that of tuberculosis, namely, a marked haziness of the joint, atrophy and destruction of the articulating sur-



FIG. 198.—Leprosy. (*Courtesy of Dr. W. B. Bowman.*)



FIG. 199.—Leprosy. (*Courtesy of Dr. W. B. Bowman.*)



FIG. 200.—Coccidoidal granuloma. (*Courtesy of Dr. W. B. Bowman.*)



FIG. 201.—Coccidoidal granuloma. FIG. 202.—Coccidoidal granuloma. (*Courtesy of Dr. W. B. Bowman.*)

faces with no bone production. The infection extends into the bones adjacent to the joint, and here we get the same destructive process as in the joint with no new bone production. The bony involvement is generally more extensive than in tuberculosis but the *x*-ray picture is so nearly like that of tuberculosis that it is impossible to differentiate these lesions.

MINERAL POISONING. Occasionally one sees bone infections resulting from mineral poisons, for example, phosphorus poisoning where the mandible is involved. The changes in this instance are the same as in chronic pyogenic infections.

SPECIAL INFECTIONS. Besides the types noted above, we have bone infections which are limited to certain bones having specific functions, such as abscesses confined to the alveolar portion of mandible and maxilla surrounding the apices of teeth, or to the destruction of mastoid cells in mastoiditis. These infections are not within the scope of this book and will not be discussed.

We have emphasized the fact that our diagnosis is based upon bone destruction and production arising from the infection. If any other cause has been added to the infection, particularly surgical interference, we may draw erroneous conclusions, because our apparent bone destruction as seen upon the plate may not be due to disease, but may be the result of the surgeon's curette. Furthermore our new bone production may take place in the normal bone through which the surgeon has passed to reach the infection. Before attempting an *x*-ray diagnosis of any bone lesion, it is always well to know whether there has been surgical interference. One should be extremely guarded in such cases in making a diagnosis.

CHAPTER IX

JOINT LESIONS IN CHILDREN

CHAPTER IX

Joint Lesions in Children

THE lesions treated of in this chapter are confined to the first age period. The vast majority of joint lesions occurring in this period can be classified in three groups:

1. Those occurring from birth up to three years.
2. Those occurring from three years up to seven years.
3. Those occurring from seven years up to the second age period.

In the first class, during the period from birth up to one year, tuberculous infections are so infrequent that they can be practically disregarded. After one year, while tuberculous infections occur, they do not begin to appear with any degree of frequency until the third year. In the same way acute epiphysitis, while probably more frequent than tuberculosis, follows approximately the same course.

In this group, especially during the first two years, joint changes are very largely confined to the lesions arising from rickets, congenital lues and scurvy.

In the second group period the three most common lesions of the first group have practically disappeared, to be replaced by the lesions of tuberculosis and acute non-tuberculous infections.

The third group period is subject to the same lesions as group two, with one other added, namely, Perthes's disease. In the latter part of this group period, just before puberty, Perthes's disease ceases to occur, and tuberculosis is the predominant lesion.

It must be remembered that this age classification is only approximate, and the periods may easily overlap.

Rickets. Now the changes that take place with the lesions in the first group period will be considered. Since rickets is by far

the most common of all joint lesions it will be taken up first. Its lesions must be studied from two standpoints; first, the changes that affect the joint, and secondly, those affecting the bones, which may be considered as remote or constitutional changes.



FIG. 203. FIG. 204.

FIG. 203.—The saucer-shaped expansion of the epiphyses of the radius and ulna in rickets, and bowing of the shaft due to softening of the bone.

FIG. 204.—The saucer-shaped expansion of the epiphyses in rickets.

In the joint changes the first thing to be noted is that the lesion does not involve one joint alone but is invariably multiple. Fluid may be present, and there is more or less swelling of the periarticular tissues. The cartilaginous surfaces of the joint are intact, but there is marked disturbance of the epiphyseal line. The epiphyseal line becomes softened and has a tendency to spread out, so that the bone at that point is

actually wider than normal. There is slight condensation of the calcium salts at the epiphyseal line, and the end of the bone has an inverted saucer-shaped appearance, Figs. 203 and 204. The epiphysis proper does not seem to be disturbed; the changes are confined entirely to the epiphyseal line. This saucer-shaped





FIG. 205. FIG. 206.

FIG. 205.—Showing the saucer-shaped epiphyses of tibia and fibula with atrophy of the bones and no periostitis.

FIG. 206.—Since the pressure is not perpendicular to the epiphysis of the upper end of the femur the rachitic changes there are atypical.

expansion is most marked where the stress and strain is directed perpendicular to the epiphyseal line, as in the epiphyseal changes in the tibia and radius, Fig. 205. In the hip and shoulder, where the stress and strain are not directed perpendicular to the epiphyseal line the changes are not so typical, Fig. 206.

Since these joints are swollen and painful, there is limitation of motion, and consequently we get a general atrophy of the bones, not only due to disuse but also due to the fact that the disease is a nutritional one. When the attack is especially severe and of long standing the atrophy becomes so extreme that the bones lose almost all of their inorganic salts and become so weakened that the slightest trauma will produce fractures. In severe cases multiple fractures are quite common, Fig. 207. The writers have seen one case in which there were eighteen fractures of the long bones, one bone, the femur, having four distinct fractures. On account of the extreme atrophy we do not as a rule have periostitis, though occasionally it may be present to a slight degree. Care must be taken not to mistake this condition for osteogenesis imperfecta. In this disease the marked atrophy and the multiple fractures are present but the point of differential diagnosis rests upon the fact that there are no joint and epiphyseal changes.



FIG. 207.—The marked atrophy of the bones in rickets is a frequent cause of multiple fractures, where there is no history of trauma. Note the fracture in the radius and ulna with some callus formation. In this patient all the long bones had sustained fractures.

In rachitic conditions it is always well to examine the chest, as in typical cases the rosary at the ends of the ribs will be found, due to the same saucer-shaped expansion of the epiphyseal line of the ribs. We have spoken of the softening of the bones due to the absorption of the calcium salts. This

same change takes place at the costochondral region and produces secondary changes in the bones that materially



FIG. 208.—Atalectatic strips of consolidation parallel to the sternum in rickets resulting from the sinking in of the costochondral portion of the ribs.

affect the pulmonary structures. With the softening at the costochondral region, coupled with muscular pull, the atmospheric pressure on the outside and negative pressure within the thorax, the sternum and cartilaginous portions of the ribs sink in and produce the typical "pigeon breast" of rickets. The deformity may be so marked that the depressed bony

structure may actually rest upon the roots of the lungs and interfere materially with the proper aeration of the lung fields. It is not uncommon in cases of marked rickets to see a narrow band of consolidation just beneath the costochondral junction on each side of and parallel to the sternum. This consolidation is in reality an atelectatic strip (Fig. 208) of lung produced by pressure and when this is present to a marked degree the result is generally fatal. In less severe cases faulty aeration is quite a factor in the production of various pulmonary lesions. With proper treatment the epiphyseal changes clear up, but there is frequently left behind a line of slightly condensed lime salts extending across the shaft of bone due to faulty calcification of the epiphysis. In a number of cases four or five such lines, parallel to each other and only a short distance apart, have been observed, indicating that there had been that number of acute exacerbations in the course of the disease, Fig. 209. With the softening of the bones deformities ensue. The most characteristic of these are the pigeon breast deformity and the saber chin. The deformities, of course, persist and do not clear up.

To summarize the chief points to be observed in rickets:

1. Disease occurs in the very young, especially during the first two years.

2. The joint is intact, the epiphyseal line softened, and spread out (saucer shape).

3. Atrophy is present with frequent occurrence of fractures.

4. Periostitis generally is absent, though occasionally present.

5. Marked pulmonary changes are present.

6. Deformities of the bones are noticeable.

7. No subperiosteal hemorrhages exist.

CONGENITAL LUES. Just as in rickets, congenital lues is accompanied by bone and joint changes. The joint involvement is generally multiple, and there is periarticular swelling. The cartilaginous surfaces of the joint are intact, but as in rickets there are marked disturbances in the epiphyseal zone

region. Here the changes take place partly in the epiphyseal line and partly upon the diaphyseal side of the epiphyseal line. There is, however, no softening or saucer-like expansion of the epiphyseal line. Instead, the process is distinctly





FIG. 209. FIG. 210.

FIG. 209.—The dark line about one inch above the lower epiphysis of the tibia showing that there was a rachitic condition present when the epiphysis was at that point. This dark line was due to faulty calcification at the time of the lesion.

FIG. 210.—Destruction of the upper end of the tibia and fibula, due to congenital lues. The diaphyseal portion of the bone, just beneath the epiphysis, is involved. Note that there is no spreading out of the epiphysis.

a destructive one. There are localized areas of softening and destruction just beneath the periosteum at the point where the periosteum stops and the cartilage of the joint begins, Fig. 210. These areas of destruction are circumscribed and

punched out, suggesting the appearance of having been bitten out by a rongeur forceps. They start at the epiphyseal line and extend back into the bone proper. The epiphyseal line in the undestroyed portions appears perfectly normal.



FIG. 211.—Note the marked periostitis generally present in congenital lues.

Atrophy is generally absent; therefore, fractures seldom occur. Luetic infections nearly always produce new bone, so that there is practically always more or less periostitis, Fig. 211. Since there is no softening of the bones, deformities do not occur in the long bones; and for the same reason, deformities are absent in the chest, hence there are no pulmonary complications.

To summarize the chief points characteristic of congenital lues:

1. The disease occurs in the very young, especially during the first two years.

2. The joint is intact; there is no spreading out of the epiphyseal line, but gouged out areas are present in the diaphysis.

3. No atrophy takes place, consequently fractures occur but seldom.

4. Periostitis is practically always present and generally excessive.

5. No pulmonary changes are present.

6. No deformities are present.

7. No periosteal hemorrhages exist.

Since rickets and lues are seen so commonly in the lower walks of life, the two conditions are frequently associated in the same individual, and it is often difficult to determine



FIG. 212.



FIG. 213.

FIG. 212.—Trümmer zone of destruction behind the epiphyseal line seen in scurvy.

FIG. 213.—Note the elevation of the periosteum with an organized hemorrhage beneath so frequently seen in scurvy. A Trümmer zone is also present.

from an *x*-ray plate which is the predominating factor in the case.

SCURVY. As in lues and rickets, this disease is always characterized by joint and bone changes. The joint involvement is generally multiple. Periarticular swelling is usually present. The cartilaginous surfaces of the joint are intact, but as in rickets and lues there are marked disturbances in the zone of the epiphyseal line. All the changes, however, take place upon the diaphyseal side of the epiphyseal line. The epiphysis and epiphyseal lines are intact, no changes taking place at all in these two structures. There is no saucer-shaped expansion of the ends of the bone as in rickets, nor

localized areas of softening and destruction as in lues. Just behind the epiphyseal line at a distance of three or four millimeters is what looks like a second epiphyseal line, in reality



FIG. 214.—A large hemorrhage beneath the torn-up periosteum which is just beginning to organize.

a band of localized destruction about two millimeters in diameter extending through the entire bone and parallel to the epiphyseal line. The edges of this band are denser than normal bone and give the appearance of eburnated bone, being due probably to condensation of calcium salts. This band (Trümmer zone) produces an appearance on the plate as if a surgeon

had operated and removed a narrow cross section of bone, Figs. 212 and 213. Atrophy may be present, but as a rule the bone texture appears normal. Fractures are infrequent.



FIG. 215.—Huge ossifying hematoma in scurvy. Note the ossification in the periosteum with organization of the hemorrhage. This tumor was so dense that it was mistaken for a sarcoma. The *x*-ray examination showed it to be an ossifying hematoma.

Periostitis is practically always present, and it is quite common to have hemorrhages beneath the periosteum. It has already been mentioned that in children the periosteum is but loosely attached to the shaft of the bone but is firmly adherent at the

epiphysis; consequently when hemorrhages occur the periosteum is very easily elevated and the hemorrhage surrounds the entire bone, frequently extending to the epiphyseal line. In the beginning this cannot be demonstrated upon a plate, but soon organization takes place and calcium is deposited not only in the periosteum but also in the blood clot. In excessive hemorrhage the pressure may be so great as actually to dislocate one of the epiphyses.

When organization of the clot has taken place the mass becomes so hard and dense that the condition may be mistaken for sarcoma, Figs. 214 and 215. The writers have seen two such instances where amputation of the leg was advised, and the true nature of the condition was only disclosed by the *x*-ray examination. The mistake in diagnosis may occur especially after the acute scorbutic symptoms have subsided. The *x*-ray, however, is so definite that once seen a mistake in diagnosis will rarely be made.

To summarize the chief points:

1. Disease occurs in the very young, generally in the first and second years.

2. The joint is intact. The epiphysis and epiphyseal line are not disturbed, but a destructive zone is formed just behind the epiphyseal line.

3. Atrophy is occasionally present. Fractures are infrequent; the epiphysis is occasionally dislocated by hemorrhage.

4. Periostitis is practically always present.

5. No pulmonary changes are present.

6. No deformities but occasional tumor masses are present due to hemorrhage.

7. Subperiosteal hemorrhage is frequently present.

It will be noted that these three diseases, namely, rickets, congenital lues and scurvy, all occur at approximately the same age, that all have multiple joint involvement, and that the joint surfaces are intact. The differential points in diagnosis depend upon changes in and around the epiphyseal line and the character of the periosteal changes.

In rickets the changes are confined to the epiphyseal line; in lues the epiphyseal line and bone behind it are involved; while in scurvy the epiphyseal line is intact and all the changes, comprised in a zone of destruction, take place just behind the epiphyseal line.

In rickets there is seldom periostitis; in lues there is marked periostitis, while in scurvy the periostitis is frequently accompanied by subperiosteal hemorrhages. In rickets we have atrophy and frequently multiple fractures; while in lues and scurvy there is generally but slight atrophy, and there are no fractures.

The changes that have been described apply only to untreated cases. When certain agents are administered in rickets, notably cod-liver oil, as has been pointed out by Howland and Park, there are marked metabolic changes not only at the epiphyseal line but also in the periosteum. The calcium salts are deposited in the cartilaginous spiphysis. Since this is not a direct continuation of the bony otructure, but is laid down about one-fourth inch from the bone proper, the roentgenogram gives a clear cartilaginous space between, suggesting a Trümmer zone such as is seen in scurvy. The epiphyseal line, however, is still slightly curved and expanded Figs. 216 and 217. There will also be a deposit of calcium salts in the periosteum. This is not a periostitis in the ordinary sense, since it is not an inflammatory process, but must be looked upon as an atypical periosteal reaction, Fig. 218.

As the close of this group period is approached, tuberculosis and acute epiphysitis become more frequent and practically supplant rickets, congenital lues and scurvy in the second group period.

Tuberculosis. In this infection the lesion is generally confined to one joint. As has been so frequently noted, bone destruction and production are essential for the determination of a lesion; consequently there is an indeterminate stage in tuberculosis when destruction has not begun and when only some fluid and periarticular swelling of the joint have appeared.

This change is not pathognomonic, as exactly the same condition may result from a ligamentous tearing. The roentgenograms of the joint, however, soon become very hazy and indistinct, and this is quite typical of a tuberculous lesion. The cartilage is gradually destroyed; and while cartilage





FIG. 216. FIG. 217.

FIG. 216.—This case simulates scurvy in that there is an apparent Trümmer zone present. The saucer-shaped expansion shows that this is a case of rickets and this irregular deposition of salts in these epiphyses is due to cod-liver oil feeding.

FIG. 217.—Rickets after cod-liver oil feeding. Note the dense deposition of lime salts at the epiphyses.

cannot be demonstrated by means of the *x*-ray, it can be determined that destruction of cartilage has taken place by the fact that the joint space has become narrowed. The disease, having destroyed the cartilage, penetrates the bone, and the bone just beneath the cartilage becomes irregular and worm-eaten. Both articulating surfaces are involved; on account of the pain the joint does not function, and atrophy results, in-

creasing with the duration of the disease, Fig. 219. The destruction of the ends of the bone is by direct extension of the infection, never by metastases to other parts of the bone leaving normal bone between. As the disease progresses there is frequently subluxation or marked distortion of the joint.



FIG. 218.—Healed rickets after cod-liver oil feeding. Note that in these cases there is invariably calcification of the periosteum which does not indicate an inflammatory process such as is seen with infection.

When the disease subsides and repair takes place, unlike other infections, there is seldom new bone production. Instead, nature replaces the destroyed areas by fibrous tissue, and when ankylosis ensues it is generally fibrous in character. When, however, such a joint goes on to sinus formation, then a different condition takes place. Since these sinuses extend

through the skin, a new channel is opened for infection, by means of which some pyogenic organism generally reaches the joint. After such an infection takes place and subsides we then have bone production, sometimes so extensive in character as completely to mask the changes that are char-



FIG. 219.—Tuberculosis of the knee joint showing marked hazing of the joint and atrophy with some destruction of the epiphyses. In tuberculous joints the diseased portion is always indistinct, due to the character of the disease, and the swelling of the soft tissues in marked distinction to the detail discernible in the bone above and below the joint.

acteristic of tuberculosis. It is important to remember, then, that mixed infections will produce new bone in an infected tuberculous joint.

Cases of tuberculosis of the bones have been discussed in the chapter on osteomyelitis.

ACUTE EPIPHYSITIS—NON-TUBERCULOUS (Fig. 220). In the early stages of this infection, as in tuberculosis, swelling and fluid are present; at this stage it is impossible to differentiate the two conditions. As the disease progresses destruction appears, but generally the picture of the joint is sharp and



FIG. 220.—Non-tuberculous infection starting in the shaft of the femur and extending to the hip joint. Note the new bone production. The original sinus in the shaft is injected with bismuth. New bone production is not seen in a straight tuberculous infection.

clean-cut, not hazy and indistinct as in tuberculosis. As a rule the lesion is confined to one joint, though it may be multiple and the destruction is more rapid and the direct extension more irregular. There appear frequently focal spots of disease in the bone not directly connected with the primary infection of the joint. As the disease subsides there is marked production

of new bone, and the ankylosis is practically always bony in character. When such a joint is examined some time after the lesion is cured, one is struck by the extensive bone production. In this lesion, wherever cartilage is destroyed the replaced tissue is always bony in character, while in tuberculosis the destroyed area is replaced by fibrous tissue.



FIG. 221.—Juvenile deforming osteochondritis. (*Perthes's disease.*) Early case showing slight flattening of the epiphysis and slight thickening of the neck of the femur.

To briefly summarize: It will be noted that in the early stages of these two lesions it is frequently impossible to make a differential diagnosis, but when the stage of repair has been reached the presence of marked production of new bone indicates that the infection was pyogenic and not tuberculous.

In this and the succeeding group periods joints will occasionally be seen in which there is swelling and fluid. Repeated examinations of such cases will show that there is never any

destruction of the joints. They will eventually return to normal. These must be looked upon as the result of some very low grade infection, or when a single joint is involved it may be the result of some slight unrecognized trauma.

At the end of this second group period and also during the first part of the third group period we have a new condition



FIG. 222.—Juvenile deforming osteochondritis. (*Perthes's disease.*) Advanced case showing marked flattening and lamination of the epiphysis and thickening of the neck of the femur. There is no hazing of the joint as is seen in tuberculosis.

which was first described by Legg of Boston and later in more detail by Perthes. The disease is now known as Perthes's disease.

PERTHES'S DISEASE OR JUVENILE DEFORMING OSTEOCHONDRITIS. In Perthes's original communication he called attention to the fact that the disease was limited to the hip joint. So far this is the only joint known to be involved; if the condi-

tion does involve other joints it has not yet been recognized, Figs. 221 and 222.

This condition is of peculiar interest in that it affects the hip joint just at the age when tuberculous infections are so frequent. Its clinical signs and symptoms are also closely akin to those of tuberculosis, but it does not react to tuberculin. The recovery is more rapid, and since there is but little destruction, as compared with tuberculosis, the hip returns more nearly to normal. What limitation of motion remains is the result of mechanical changes in the joint.

It is frequently impossible to make a diagnosis from the clinical picture, but in typical cases the *x*-ray appearance is pathognomonic and when once seen can always be recognized.

It is much more common in the male than the female, and seems to occur most frequently between the ages of seven and twelve. There is no hazing and clouding of the joint as in tuberculosis; on the contrary all bone detail is clean-cut and sharp. The epiphysis is not eroded nor worm-eaten.

The epiphysis seems to have softened and become flattened out as if from pressure. The cartilage is intact, but the compression of the epiphysis makes it seem denser than normal, as if there were increased deposition of calcium salts. The epiphysis is occasionally separated into several centers as if broken by pressure. The neck frequently seems broader than normal, and there may be slight coxa vara present, as if the neck had softened and was bent. The lesion apparently confines itself to the epiphysis and does not involve the acetabulum directly though changes in the shape of the epiphysis of the head of the femur may later mechanically alter the acetabulum. One or both hips may be involved.

The etiological factor in this disease is unknown; apparently a condition of lessened resistance is brought about with resulting secondary changes due to weight.

Tuberculosis and acute infections continue throughout the third group period. With the union of the epiphyses adult life is reached, embracing that great group of rheumatoid affections which will be dealt with in another chapter.

CHAPTER X

JOINT LESIONS IN ADULTS

CHAPTER X

Joint Lesions in Adults

BEFORE dealing with the pathological processes involving a joint it is well to remember its parts.

A normal joint consists of four structures, the articulating surfaces composed of cartilage, synovial membrane, ligaments and synovial fluid. There is neither free bone nor periosteum in a joint. The diagnosis of joint conditions is accomplished by carefully considering the changes in one or more of these four parts. The cartilage is probably the most important; it is the involvement of this structure which leads to chronic conditions. Certain types of infection involve only the synovial membrane, with secondary changes as to the amount of synovial fluid. In this type of joint infection a return to the normal condition may reasonably be hoped for, while when the cartilage is involved the return to a normal condition depends largely upon the severity of the infection.

The *x*-ray appearance is frequently so different in the same condition that confusion may arise unless the following important point is kept constantly in mind, namely, that the appearance of the joint changes according to the stage of the disease obtaining when the examination is made.

Every infection may be divided into the stages of: (1) onset, (2) maximum intensity and (3) repair. It will be readily seen that the *x*-ray appearance must vary in each of these stages. This can be illustrated graphically. Let the line *A–B* represent a normal joint, and consider the curved line *C–D* as representing an infection where the cartilage is not involved. At *D* this line merges with the line *A–B* and the joint has returned to normal. Now let the curved line *E–F–H* represent the curve of some acute infectious process. It starts from the normal line *A–B* at *E*, and at *F* it reaches its nearest point to

the normal line; from there, *F–H*, it runs parallel to *A–B*. This indicates that permanent changes have taken place in the joint and that it will never return to normal. The infection has disappeared, but there remains behind a more or less permanently impaired joint depending entirely upon the severity of the infection. In the diagram the numeral 1 represents the stage of onset, 2, the stage of maximum intensity, and 3, the stage when the infection has subsided and repair is taking place. If an arthritic infection is considered from the standpoint of these three stages there will be no difficulty in understanding why the *x*-ray changes are varied in the same infection.

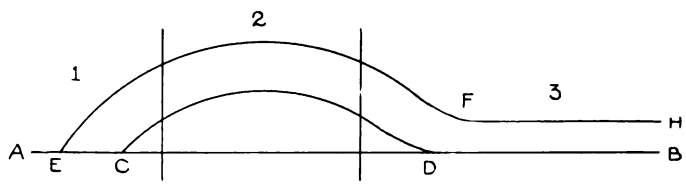


FIG. 223.—Graphic illustration of the stages of bone infection: (1) onset, (2) maximum intensity and (3) repair.

CHANGES INDICATING AN ARTHRITIC CONDITION. Arthritic conditions may be divided into two classes. First the acute polyarticular infections where there are no permanent changes and the joint returns to normal; second, that large group of so-called chronic arthritides known as arthritis deformans.

Attention has been repeatedly called to the fact that *x*-ray evidence depends upon variations from the normal, and that the two great factors producing variations from normal are bone destruction and production. In a normal joint there is no free bone, so at first those changes must be considered which may be termed indirect evidence, as the normal factors entering into the structure of a joint are not directly demonstrable upon an *x*-ray plate. The first change that takes place is swelling of the synovial membrane. In the majority of the joints the flesh and skin outline will indicate this by the enlargement of the soft tissues. With this inflammatory change of the synovial membrane, fluid will form; in most joints one can

infer this by the position of the part and the swelling around the joint; in the case of the knee the riding patella will be seen. If this infection is severe the next point of attack will be the cartilage. In this case also this structure is not demonstrable by the *x*-ray and again the condition must be determined by indirect evidence, that is the width of the joint space. If the cartilage is uniformly eroded, the bones, because of the muscular pull, will be closer together; thus the joint space will be narrowed. The infection may go on to complete destruction in certain areas and attack the bone beneath. The moment the bone becomes involved there is direct evidence of destruction that can be demonstrated upon an *x*-ray plate.

Cartilage reproduces itself only to a limited degree, and if a portion is completely destroyed down to the bone, nature replaces it either with fibrous tissue or with bone. It is in this manner that what might be termed "free bone" in a joint is developed.

Besides the two main factors of destruction and production of bone, atrophy both of bone and muscle must also be taken into consideration. The changes just described do not take place immediately, but extend over a period of time. For that reason it is wise to study the joints from the three stages already mentioned. In a previous chapter the lesions of joints up to puberty were grouped and described under certain age periods. Here, again, it will be shown that age plays an important part in rheumatic infections. These will, therefore, be considered under the following groups: (1) those occurring between twenty and forty years, and (2) those occurring after the fortieth year.

Acute Polyarticular Rheumatism. This disease (Fig. 224) is seen most frequently between twenty and forty years of age and, as its name suggests, involves more than one joint. In this lesion the *x*-ray findings are rather indefinite. In the first stage the synovial membrane and periarticular tissues are swollen and there is an increased amount of fluid in the joint. In this stage it is too early to see cartilaginous changes,

and consequently there is no necessity to look for them. This first stage lasts for a week or ten days; in that short period of time no atrophy will be present. In the second stage of the disease cartilaginous destruction might be expected. This, however, does not occur in acute polyarticular rheu-

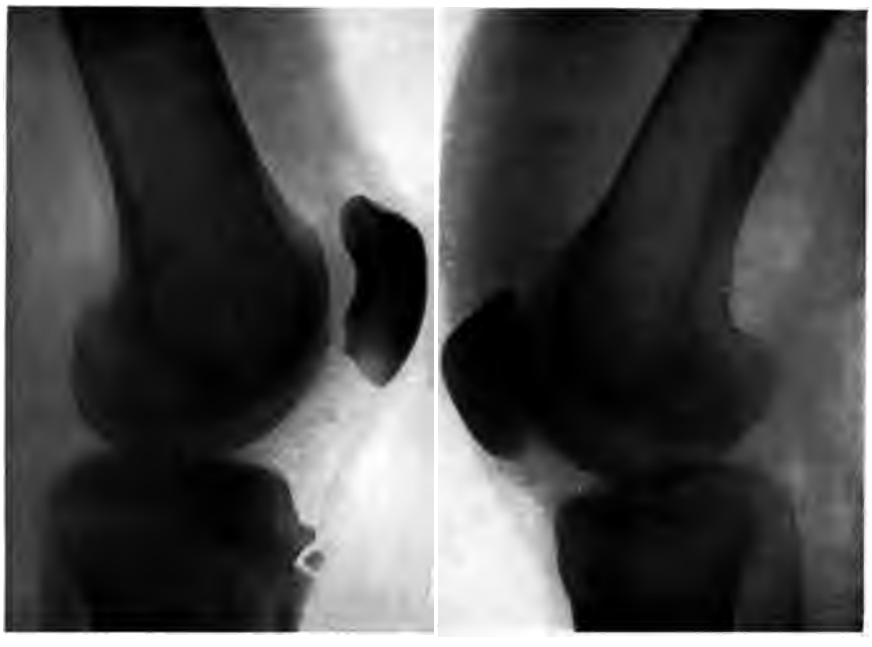


FIG. 224. FIG. 225.

FIG. 224.—Acute polyarticular arthritis, showing the riding up of the patella indicating fluid. There is no involvement of cartilage or bone.

FIG. 225.—Infectious arthritis in the first stage, showing swelling of the soft tissues and beginning fluid formation. In this stage it cannot be differentiated from acute polyarticular rheumatism or trauma to the joint.

matism because under treatment the swelling disappears. In this disease the second stage is of brief duration, consequently, there is no atrophy. The third stage, or the stage of repair, is reached in about three weeks after which the joint is again normal.

The only changes, then, seen throughout the disease are

swelling and fluid in the joint. No atrophy is present, as the duration of the disease is too short. These changes are not pathognomonic, as exactly the same *x*-ray changes are seen in a traumatic injury to a joint, or in the first stage of an infectious arthritis, which will be described later.

CHRONIC ARTHRITIS. In considering the chronic rheumatic infections great difficulties are encountered. The classifications of the various types under this head are very unsatisfactory, and then too these chronic rheumatisms have many names for the same condition. For example, the terms chronic rheumatism, arthritis deformans, rheumatoid arthritis, osteoarthritis, spondylitis, etc., are used indiscriminately by different individuals to mean the same condition. There is no classification which is absolutely satisfactory, but that adopted by Goldthwaite many years ago is, taken as a whole, probably the best. Of course many take exception to this classification and do not agree with it in its entirety; but its simplicity and the great number of cases which can be classified according to it make it seem to be generally the most useful.

He divides chronic arthritis into:

1. The infectious type. This includes tuberculosis, gonorrhea, lues, pneumococcic infections, etc., and those types of arthritis which have the same clinical manifestations, but where the exact etiological factor is unknown.

2. Those cases designated as atrophic arthritis. It is questionable whether this group can be looked upon as a distinct entity.

3. Hypertrophic arthritis.

Infectious Arthritis. This group does not seem to be limited to any age period, and is just as prevalent in the second as in the third age period. It is true that certain acute infections, such as gonorrhea or tuberculosis, are much more common in the second age period.

From an *x*-ray standpoint, infectious arthritis presents three different appearances according to the stage of the

disease at the time of the examination. In the first stage, (Fig. 225), at the onset of the infection, the plate shows distinct swelling of the soft tissues around the joint with increase of the synovial fluid. There is no cartilaginous destruction, evidenced by the fact that the joint spaces are of normal width. Since the cartilage has not been involved, of necessity there can be no bone change. There is no atrophy, as sufficient time has not elapsed to bring about this condition.



FIG. 226.—Second stage of infectious arthritis, showing destruction with narrowing of joint space, but no new bone production.

This stage gives the same *x*-ray appearance as an acute polyarticular rheumatism or an injury to the joint without fracture, because in these conditions fluid and periarticular swelling are also present, and no cartilaginous changes appear.

In the second stage the disease probably reaches its maximum intensity, and in time the following changes take place in the joint and bone. Sufficient time has elapsed for the joint to become more or less immobilized either from pain or treatment, consequently atrophy of the bones forming the joint appears.

The swelling and fluid are still present. The cartilage in this stage has become involved and may be more or less destroyed according to the severity of the infection; this is evidenced by the fact that the joint space is narrowed, Fig. 226. The infection may have localized sufficiently at one part of the joint to destroy all the cartilage and actually involve the bone beneath; in this case any bone change can be easily seen upon the plate. While the soft tissues around the joint are still swollen there may be atrophy of the soft tissues above and below the joint. This is particularly true in long-standing infections, such as tuberculosis. This second stage is variable as to length of time, and the same changes persist as long as the infection is active. The atrophy, however, becomes greater the longer this acute stage exists. There is no production of new bone, as this is a repair process and cannot take place during the height of the disease.

With the subsiding of the infection the third stage, or stage of repair, is ushered in. Here, too, the picture is slightly different depending upon whether the examination is made at the beginning or at the end of this stage. The swelling and fluid have disappeared, the atrophy is still present, and will only disappear when the joint begins to function again. The destructive process, however, has ceased and repair now takes place. Unfortunately the cartilaginous surfaces which are so easily attacked have but little recuperative powers in themselves. Cartilage only reproduces itself to a limited extent, and, in areas where it is completely destroyed, it does not fill in; so nature has to fall back upon two substitutes: the destroyed area is either filled in with fibrous tissue, or the raw, exposed bone proliferates, filling up the destroyed area. This overgrowth is spoken of as an exostosis, Fig. 227.

The extent of the overgrowth of bone is dependent entirely upon the extent and severity of the infection. Where two opposing articulating surfaces are denuded of cartilage, the resulting new bone formation from both surfaces may unite and produce an actual bony ankylosis. It is interesting to

note that the infection or its toxin shows a marked predilection for the ligamentous attachments, and the first bone proliferation is found at these points.

When the joint has recovered as far as it is possible there is absence of swelling and fluid, atrophy has disappeared, and



FIG. 227.—Third stage of infectious arthritis. The infection has now subsided and repair is taking place. The damaged cartilage is replaced by new production of bone and the joint is permanently damaged according to the severity of the infection.

there is the marked formation of exostoses or actual bony ankylosis depending entirely upon the severity of the lesion.

While this is, relatively speaking, a rough picture of what happens in infectious arthritis there are variations depending upon the particular type of infection.

The foregoing analysis applies to the subdivisions under infectious arthritis of known etiology. The question naturally

arises, do any of these conditions give such definite *x*-ray changes that the roentgenologist can actually determine the specific type of infection with which he is dealing. Unfortunately the majority of them give the same *x*-ray indications. Some, however, such as tuberculosis, gonorrhea and lues, give fairly constant appearances, so that they can often be recognized.

Tuberculosis. (Figs. 228 and 229.) In this lesion there is at first the swelling and fluid which is characteristic of most joint lesions. Soon, however, the joint becomes hazy and indistinct. In fact, it suggests a very poor plate, but in this same plate it will be noted that the bones adjacent to the affected joint come out sharply and distinctly. Only the joint itself is hazy and indistinct. This is probably due entirely to the changes in the synovial membrane. The joint spaces have become narrowed, owing to the destruction of the cartilaginous surfaces, and upon close inspection of the plates it will be found that the bone contour, that is, the bone beneath the cartilage, is very indistinct, irregular and worm-eaten. Fluid and periarticular swelling are still present, and if the lesion is of some duration the plate shows the spindle-shape joint of tuberculosis due to atrophy of the soft tissues above and below the swollen joint. There is also marked atrophy of the bones, due to deposition of fat in the bone.

When the stage of repair takes place the haziness and indistinctness disappear, but there remain behind the irregular worm-eaten ends of the bones more or less completely denuded of cartilage. Theoretically the repair process should take place with replacement of cartilage, but unfortunately this structure has but little recuperative power. Bone and fibrous tissue are the only other substances that nature has at her disposal in this region, and consequently the denuded areas in this type of disease are replaced entirely by fibrous tissue. For some reason but little bone production is encountered in tuberculous joints. When ankylosis takes place it is almost entirely fibrous in character. Since the disease is of

long duration and the joint more or less fixed, the muscular pull plus the lesion in the joint produce marked subluxations. Since these joints are so frequently untreated, the formation of sinuses is quite common, and, just as in children's joints, a secondary infection ensues. In these mixed infections marked new bone formation is seen. Tuberculosis of the joints is largely a disease of childhood and of the first twenty years of adult



FIG. 228.—Tuberculous arthritis of the wrist, showing swelling, hazing and destruction of cartilage and bone.

life, though occasionally one sees an acute tuberculous joint after forty years of age. Practically any joint can be involved; tuberculosis of the shoulder joint, however, is uncommon in childhood and early adult life.

Caries Sicca. After forty years of age one form of tuberculosis develops whose favorite site, curiously enough, is the shoulder joint, Fig. 230. Caries sicca presents a totally different *x*-ray picture. There is never any fluid, swelling or hazing. The cartilage is destroyed, the bone beneath is worm-

eaten, and frequently gouged-out areas are present just beneath the joint surfaces. These changes are sharp and clean-cut in outline. The striking features are the extreme atrophy of the bones and soft tissues. The soft tissues have become so atrophied that the curve of the shoulder disappears completely, clinically simulating a subluxation. The synovial membrane has also contracted; consequently, the head of



FIG. 229.—Tuberculous arthritis of the knee, almost healed. Notwithstanding the extensive destruction, the joint is clear cut as the hazy indistinct joint is only present during the active process of the disease.

the bone is pulled high up in the glenoid fossa. It is frequently difficult to get a good plate on account of the extensive bone atrophy.

Gonorrheal Arthritis. Gonorrheal arthritis (Fig. 231) does not present a distinct *x*-ray appearance. The roentgenogram varies according to the severity of the lesion. Only in the knee-joint are there fairly definite characteristics. For

some reason in that particular joint the greatest severity of the lesion manifests itself just beneath the patella, and early bone ankylosis occurs. This condition may result from other infections, but according to our present knowledge ankylosis between the patella and femur is nearly always looked upon



FIG. 230.—Caries sicca of the shoulder joint. Note the marked atrophy of the bone and soft tissue with marked bone destruction. This is a lesion of the third age period and since there is no swelling of the soft tissue and fluid, we do not have the characteristic hazing of the joint seen in the other form of joint tuberculosis.

as of gonorrheal origin unless the identity of some other organism can be definitely established. The usual changes in a gonorrheal joint are those of acute destruction with new bone production and frequent ankylosis. This lesion is by far the most frequent in early adult life.

Luetic Arthritis. Luetic arthritis (Fig. 232) shows marked periarticular swelling, thickening of the synovial membrane and fluid in the joint. This type does not go on to cartilaginous destruction, consequently when the disease subsides it leaves a perfectly normal joint. The *x*-ray appearance, as far as



FIG. 231.—Gonorrheal arthritis with beginning changes of the under surface of the patella, and the tibia and femur. The changes are similar to other types of infections of the joint, except that in gonorrheal arthritis there is patella involvement, although this may not always be present.

the joint is concerned, is identical with that of an acute polyarticular rheumatic or a traumatic joint. Fortunately in the majority of cases this type of arthritis is generally accompanied by bone change, and while this change is not in the joint itself, it is fairly characteristic, and may be recognized

as a small area of periostitis formed just at the chondro-periosteal junction. This periostitis, in conjunction with peri-articular swelling and fluid in the joint, is fairly characteristic of a luetic lesion. This lesion is seen most frequently in early adult life. The question will immediately be raised that peri-



FIG. 232.—Luetic arthritis with periosteal changes close to the joint.

ostitis occurs with other forms of acute joints. This is perfectly true but the differential point is that in such joints there is always destruction in the joint itself while in syphilis the periostitis is associated with no joint destruction.

The remaining forms of infectious arthritis do not give any definite *x*-ray appearances but vary according to the type and virulence of the infection.

Atrophic Arthritis. This lesion (Fig. 233) is generally seen in early and middle age, between the ages of twenty and forty. There is marked atrophy of both the soft tissues and bone. The joints are frequently partially subluxated due to muscular contraction and there is partial ankylosis not due to bone



FIG. 233.—Atrophic arthritis. Note the marked atrophy of the bone and soft tissues.

but to fibrous changes and muscle contracture. The *x*-ray examination shows marked atrophy of bone and soft tissue. There is extensive absorption of cartilage and its complete destruction in certain areas, but apparently nature makes no attempt at new bone formation. From this brief description one can readily see that atrophic arthritis simulates very

closely the second stage of infectious arthritis. The differential point is that at no stage in this disease is there any fluid or periarticular swelling, while in the second stage of infectious arthritis there is both swelling and fluid. Many clinicians think that atrophic arthritis is not a distinct clinical entity, but probably an atypical manifestation of infectious arthritis.



FIG. 234.—Hypertrophic arthritis showing overgrowth of bone, exostoses and joint mice lying beneath the head of the femur.

Hypertrophic Arthritis. Hypertrophic arthritis (Fig. 234) is a disease almost invariably associated with people of middle and old age. One rarely sees this type in those under forty, and it is most pronounced in patients in the neighborhood of fifty and over, so it may be said that this is a disease of the third age period.

Attention has already been drawn to the fact that as we grow older there is a generalized atrophy of all the bones which we look upon as a normal change due to old age. Consequently, since this disease is one of old age, one would naturally expect to find this atrophic change in hypertrophic arthritis; on the contrary, however, the *x*-ray examination shows no



FIG. 235.—Note the calcified bodies (joint mice) lying in the quadriceps of the bursa. These are frequently seen in hypertrophic arthritis and cause the mechanical locking of the joint.

atrophy and apparently an increased deposition of calcium salts, so that the bone shadows are even a little denser than normal.

There is no swelling or fluid in the joint. The cartilage is destroyed in focal spots and in these areas bony exostoses

appear and there is marked new bone formation at the attachments of the ligaments of the joint. Frequently little bony bodies may be present lying free in the joint cavity, Fig. 235. In this condition these joint mice may become engaged in the joint, resulting in swelling and fluid; but this is an accidental factor, and is not the normal course of the disease. There may be ankylosis of the joint, but this is not a true ankylosis due to fibrous tissue or bone union, simply a mechanical locking of opposite engaging exostoses. This type simulates the third stage or the stage of repair in an infectious arthritis. The two conditions, however, would only be confused in old people since the hypertrophic type does not exist in the second age period. In hypertrophic arthritis there is increased density of bone, while in infectious arthritis there would be the normal atrophy of age.

Briefly to summarize the difference between these different conditions the following points are to be borne in mind:

Acute polyarticular rheumatism and the first stage of infectious arthritis may occur at the same age and are not distinguishable until the stage of destruction appears in infectious arthritis. The beginning of the third stage of infectious arthritis and the atrophic type both show destruction and no bone production and occur at the same age, but there is atrophy of both bone and soft tissue in the latter type, while there still remains some soft tissue swelling in the former type.

The infectious arthritis of old age can be distinguished from the hypertrophic type by the fact that with the former there is the bone atrophy of old age while with the latter there is increased density of bone.

Atrophic arthritis is seen largely in the second age period, hypertrophic arthritis in the third age period, while infectious arthritis occurs in any age period.

ARTHRITIC CHANGES DUE TO AGE. Great care must be taken not to confuse the slight arthritic changes which are always present in old people with an acute active process. It

has been definitely established that practically all of us when we reach the age of forty-five show small exostoses in and around the joints, particularly where the ligaments attach, and yet there may be no clinical manifestations of an arthritic process. This naturally means that there is a quiescent arthritis present, which only needs the proper stimulation to become an active process. It is in just these cases that such marked symptoms follow a slight injury—symptoms out of proportion to the extent of the injury. In this condition it must be remembered that the injury has simply lowered the resistance of the joint and has allowed this quiescent arthritis to flare up into an active process, so that the marked clinical manifestations seen in such joints are really the result of the arthritis and not of the trauma.

The writers have frequently seen cases where the ankle and entire foot have been enormously swollen following a slight injury, the swelling and tenderness being so marked as to suggest fracture of several bones; but the *x*-ray examination showed a low grade infectious arthritis indicated by small exostoses, and one or two of these might actually have been broken off. These cases always do better when treated from an arthritic standpoint rather than as an injury.

Arthritis in the Spine. These arthritic lesions produce the same changes in the spine as in other joints. There are, however, two special types of arthritis occurring in the spine, the acute spondylitis deformans, and the Marie-Strümpell type of spondylitis. Since all lesions of the spine will be taken up in a separate chapter in order that the differential points may be more clearly brought out, these two lesions will be discussed there.

Villous Arthritis. In closing this discussion upon arthritis it seems advisable to mention briefly the so-called villous arthritis, Fig. 236. An *x*-ray plate of such a joint will show a swollen and thickened synovial membrane so thick that at times the folds of the membrane can be demonstrated. The bony and cartilaginous changes are very variable, ranging

from a normal joint to the hazing and clouding of a tuberculous joint. It seems to the writers that this term "villous" is a most unfortunate one, as it describes a symptom rather than a condition. This villous change has been observed in an old, chronically inflamed traumatic joint, in tuberculosis, in infectious and in hypertrophic arthritis. It is a condition



FIG. 236.—The marked synovial thickening in villous arthritis.

brought about apparently by any long-continued irritation of the synovial membranes, consequently the term should not be used to designate a certain type of arthritis.

NON-ARTHRITIC JOINT LESIONS. Besides these arthritic conditions which we have attempted to describe, there are four lesions which involve joints and yet cannot be properly

looked upon as arthritic in character, namely, gout, Charcot joint, syringomyelia and the joints of hemophilia.

Gout. Gout is a disease of the third age period; it occurs more frequently in the male than in the female and is relatively infrequent when compared to other joint lesions. In characteristic cases where there are large deposits of urates the *x*-ray appearances are quite typical. There is periarticular swelling, and on account of pain there is immobilization of the joint, producing some atrophy. The characteristic changes are punched-out areas in the bones at the margins of the articular surfaces, Fig. 237. These punched-out areas vary in size, sometimes being so large as to involve the entire joint surface. They are generally filled with urates, and since urates do not cast shadows we get these apparent holes in the articulating surfaces.

In the very early stages of the disease there is some new bone formation in the shape of exostoses such as are seen in arthritis. In fact, at this stage of the disease the *x*-ray appearance is very similar to that of hypertrophic arthritis, Fig. 238.

In the advanced stage of the disease, particularly if a knee is involved, the destruction may be so great as to simulate a Charcot joint. The phalangeal joints of the hands and feet are most commonly involved, and give the most typical picture. When the long bone joints are involved the changes are most apt to be of the hypertrophic arthritic type.

Charcot Joint. The acute luetic joint which is looked upon as infectious in origin has already been described. While the Charcot joint is luetic in origin it does not take place in the acute infectious stage of the disease, but is secondary to changes taking place in the nervous system in the tertiary stage of the disease. It may be spoken of as neuropathic in origin. Since it is associated with the late manifestations of lues, it should not be looked for in the young; it is entirely a disease of middle or old age, that is of the third age period.

Any joint may be affected, but the knee, hip, ankle and spine are most commonly involved. Since these joints are



FIG. 237.—Showing the punched-out areas of the articulating surfaces of the joints so characteristic of gout.



FIG. 238.—Early case of gout where the typical punched-out areas have not appeared. There is definite new bone formation in the shape of exostoses, simulating the changes seen in hypertrophic arthritis.



FIG. 239.—Charcot joint showing the complete destruction of the joint with bony detritus in the bursa. Note that there is no atrophy present.



FIG. 240.—Anteroposterior view of the same joint.

weight-bearing they show the most typical changes. The knee is involved more frequently than any other joint and an *x*-ray examination of this joint gives the best picture, Figs. 239 and 240. The most striking change noted is the very extensive destruction of the joint. The condyles of the femur



FIG. 241.—Charcot joint of the ankle showing the marked periostitis of the tibia and fibula associated with extensive joint changes.

are broken off, more or less disintegrated and pushed aside, so that the lower end of the femur rests upon the tibial articulation and by pressure actually gouges it out. The bones become softened and the calcium salts are compressed so that the bone in this area is dense and much eburnated. The joint is very much swollen and there is fluid present.

The periosteum above the joint is affected so that there is

a marked periosteal reaction, Fig. 241. As one views such a joint with the marked destruction, one is forced to conclude that this extensive destruction must have taken place over a long period of time and also that on account of the great disintegration such a joint should be extremely painful.



FIG. 242.—Hemorrhagic cysts beneath the cartilage of the joint seen in hemophilia.

If these deductions were correct, namely, that the joint was very painful and that the condition had existed over a long period of time, then there would be disuse of that joint resulting in extreme atrophy of the bones. On the contrary the plate shows that the bones are really denser than normal, just the reverse of atrophy. The absence of atrophy then forces the conclusion that the joint has been in constant use,

and since this would not be the case if it were painful the conclusion is inevitable that the joint was painless upon motion. Now there are only two conditions in which disintegration of joints exists without great pain, and the most common of these is the Charcot joint.



FIG. 243.



FIG. 244.

FIG. 243.—Hemophiliac joint with the joint surfaces and the bone beyond the joint destroyed by hemorrhage.

FIG. 244.—Organization of hemorrhage in the elbow joint in hemophilia.

Syringomyelia. It must be borne in mind that the lesions of this disease are neuropathic in origin and give practically the same *x*-ray changes as Charcot joint, except that the lesions are largely confined to the upper extremities, while in Charcot joint the weight-bearing joints are the ones most commonly involved. Every case in which absence of atrophy is seen in connection with a badly disorganized joint should be

viewed either as Charcot or syringomyelia according to whether the lesion involves the upper or lower extremity. In certain cases it may be impossible to differentiate them without the clinical history.

Hemophilia. Occasionally one sees a joint giving the clinical symptoms of an infectious arthritis, yet the *x*-ray examination shows a rather remarkable joint condition. There are areas of marked destruction of the cartilage with gouged-out areas extending through the cartilage into the bone, Fig. 242. Sometimes there will be circular gouged-out areas in the bone behind the cartilage but not involving it, Fig. 243. Again there may be complete destruction of the entire surface of the joint, and a suffusion of blood into the synovial cavity which may go on to actual organization and deposition of calcium salts, Fig. 244.

The joint is hazy and indistinct and may simulate tuberculosis, but the differential points to be noted are the gouged-out areas behind the joint surfaces or the organized blood clots which do not occur in tuberculosis. When such a condition is present it is invariably the result of hemophilia.

ARTHRITIC CHANGES IN LIGAMENTS. Besides these joint conditions certain changes take place in the ligaments and bursae, which, while not involving joints, should be looked upon as arthritic in origin. In the chronic arthritic group there is always more or less calcification of the ligaments at their bony attachments, suggesting that these deposits are exostoses; but the deposits are entirely in the ligaments, Fig. 245. This is particularly true in the tendo Achillis and the quadriceps attachment to the patella. The entire crests of the ilia may be roughened from the same condition. These changes may frequently precede the joint changes and should always be looked upon as infectious in origin. There are two bursae which seem to be the favorite site for arthritic changes, (1) the subdeltoid, and (2) the bursa situated at the attachment of the plantar fascia to the os calcis. The latter condition is known as exostoses of the os calcis or painful heels.

Subdeltoid Bursitis. The subdeltoid bursa is situated just at the external side of the greater tuberosity of the humerous and about five mm. from the cortex. Calcium deposits



FIG. 245.—Trauma following upon an arthritic condition, producing marked new bone formation in the tendo Achillis. Of course, this is an exaggerated case.

take place in the walls of the bursa and not in the bursa itself. This usually brings about a very painful condition with limitation of motion. The *x*-ray examination will show



FIG. 246.—Showing deposition of salts in the subdeltoid bursa.



FIG. 247.—Deposition of calcium salts in the subdeltoid bursa.



FIG. 248.—The non-gonorrheal type of exostosis of the os calcis. It has the same structure as the bone itself.



FIG. 249.—Gonorrheal exostosis. The calcification takes place at the attachment of the planta fascia where there is a small bursa and finally unites with the os calcis.

a deposit of calcium salts just to the outer side of the greater tuberosity but not attached to it. With no history of injury the diagnosis is easy to make, Figs. 246 and 247. In a certain number of cases, however, this deposition may take place without the production of any symptoms at all. In such conditions a trauma of the shoulder will produce an acutely painful joint with limitation of motion. The *x*-ray will show these calcium deposits; but with apparently a normal joint before injury and symptoms following the trauma, it is frequently impossible to determine whether this is a subdeltoid bursitis or a small fragment of bone torn off by the attachments of the suprascapular muscles. The diagnosis sometimes can only be made by observing the course of the disease; if the symptoms do not subside with fixation of the arm the condition is probably a bursitis and not a ligamentous tearing. This condition is not seen in the young, but is most common in the third age period.

Exostosis of the Os Calcis. Two types of deposit are noted in exostosis of the os calcis, both infectious in origin. The condition is generally known as painful heels. In one type there is an exostosis springing from the os calcis at the point of the plantar fascia attachment, Fig. 248. It starts from the bone, grows outward like a stalactite, has the same structure as the bone and is a true exostosis. It is most frequent in the latter part of the second and all of the third age period.

The second type is of gonorrheal origin (Fig. 249), and the calcium salts are deposited first in the bursa at a short distance from the cortex. Then fresh deposits are subsequently placed until it becomes attached to the cortex. The deposits seem to be laid down in layers, and the mass does not have normal bone structure. It cannot be looked upon as a true exostosis. This type is seen most frequently in the second age period.

CHAPTER XI

BONE TUMORS

CHAPTER XI

Bone Tumors

BEFORE discussing bone tumors, reference must again be had to the normal bone, as a correct diagnosis depends upon variations of its constituent parts from the normal. A long bone is composed of an outside fibrous sheath called periosteum, not demonstrable by the *x*-ray in its normal condition. Beneath it is a dense compact bone known as cortex, composed of bone cells and masses of inorganic salts traversed by numerous small canals. Within is the medullary canal containing the marrow, fat, nerves, bloodvessels and lymph channels. At the ends of the bone the cortex and medullary canal merge into finely reticulated bone forming the cancellous ends, and these, in turn, are covered by cartilage forming the joint. The bone is pierced halfway between the ends by a small canal perpendicular to the shaft. The canal is the point of entrance for the nerves, blood and lymph vessels. Since the malignant cells are carried by either the blood or lymph, this nutrient canal plays a very important part in metastatic malignancy.

In the diagnosis of bone tumors the essential thing is to determine whether the growth is malignant or not. If that point can be established the surgeon will be given the information that is necessary for him to determine the character of the operation to be performed. It is, however, still better if the roentgenologist can go further and determine the exact nature of the lesions.

With the exception of the cartilage any one or all of the constituent parts of the bone may be involved by neoplastic growths. In the chapter on joint lesions it was pointed out that cartilage is quite easily attacked by an infection, but is quite resistent to tumors, and is seldom if ever involved by them.

Attention has been repeatedly called to the fact that the only direct changes that can be seen upon an *x*-ray plate are bone destruction and bone production, and that it is the variation in these two processes that yields certain data upon which to formulate a diagnostic hypothesis. In addition, as we have mentioned, there are two accessory aids of great value—age and sex. One does not expect to find carcinoma in the young. If a malignant tumor is found in the bone of a child it is known that it must be sarcomatous in origin. In the same way a malignant growth of the pelvis of a female over fifty years is probably a carcinomatous metastasis from the breast, as that tumor predominates in the female, while in a male pelvis of the same age the growth is apt to be a metastasis from the prostate, as that tumor is the most frequent in the male.

METHOD OF ANALYZING BONE TUMORS. By studying the point of origin and the character of the bone changes the writers have been led to formulate a rough outline for the analysis of these various bone tumors. Four points have been taken as the basis for classification. They are:

1. Origin of the tumor.
2. Presence or absence of bone production.
3. The condition of the cortex.
4. Invasion.

At first glance it is often impossible to determine all four of these points, but if one or two of them can be established they frequently lead to the correct diagnosis.

It must be borne in mind that these points, which have been named the cardinal points, are merely methods of approach to a diagnosis. If one such point can be established it automatically rules in or out certain tumors, and will generally aid in establishing a second point; thus eventually all four will be established, and as each point eliminates certain tumors, a diagnosis by exclusion is obtained. As an example of this method, if by the first point it can be absolutely shown that the tumor arises from the periosteum, that automatically excludes a carcinoma, as there are no primary epithelial cells in bone, and conse-

quently if an epithelial growth develops there, it must be carried in by the lymph or blood. Since these vessels enter the medullary canal by means of nutrient channels, carcinoma must of necessity have its origin in the medullary canal. Again, if it can be established that there is new bone in the tumor, that would also automatically rule out round and spindle-celled sarcomata and carcinomata.

In no lesions of the bones is it more important to have a knowledge of pathology than in bone tumors. The *x*-ray plate must not be regarded as a photograph in the ordinary sense. Medical knowledge must be brought to bear upon it. The roentgenologist must be familiar with the growth of tumors; he must know how they involve the bone and how and why they metastasize. *Never make a diagnosis of a tumor because it looks like something that has been seen before.* The diagnosis must be made upon the fundamental facts of pathology which have been translated into shadows and registered upon the plate. After the diagnosis is made, the reasoning must then be reversed, and each pathological point of the tumor in question applied to the plate, and they must fit in. If they do not the diagnosis is probably incorrect. This point cannot be emphasized too strongly.

These four cardinal points may now be examined, bearing in mind, however, that they are merely methods of approach, and that while they will help in the vast majority of cases, yet like all other methods of medical classifications they are not infallible.

Point of Origin. By point of origin is meant the position where the growth arises whether in the medullary canal (Fig. 250) or whether it springs from the cortex or periosteum, Fig. 251. The cartilaginous articulating surfaces, are not included, as tumors do not arise from them. If this can be determined one point of differential diagnosis is established. All tumors in bone must be either primary or metastatic. It has already been mentioned that there is no primary epithelial tissue in bone, and that if carcinoma is present it must, by means of the vessels



FIG. 250.—A tumor arising within the medullary canal. (Giant-cell sarcoma.)



FIG. 251.—A tumor arising from the cortex. (Fibromyxoma.)

through the nutrient canal, have lodged in the medullary canal and grown there. So carcinoma must always be metastatic in origin, and must arise in the medullary canal.

Since sarcoma is of connective tissue origin, it may be either primary in the medullary canal or metastatic in origin. It is also known that enchondromata may be either cortical



FIG. 252.—Bone production within a tumor. (Osteoma.)

or medullary in origin, while bone cysts are nearly always medullary in origin and very rarely cortical.

Periosteal and osteosarcomata are cortical in origin, as well as osteomata. Ossifying hematomata are not bone growths, yet clinically may simulate them and so they have been classed among tumors and they may be looked upon as periostial in origin. Sometimes when the growths have become very extensive it is impossible to determine their origin.

While this cardinal point limits the number of tumors, some having been excluded by their points of origin, one is still far from a diagnosis. The second cardinal point, therefore, is brought to bear upon the problem.

Bone Production. Bone production does not take place in either carcinoma or in round-cell, spindle-cell, or giant-cell sarcomata. Consequently if it can be established definitely that there is bone production within the tumor the above mentioned growths can be ruled out, Fig. 252. That in turn will again limit the tumors to osteoma, osteochondroma, periosteal, and osteosarcoma, and ossifying hematoma. Bone cysts are not bone producers, but since they are so frequently accompanied by fracture, bone production laid down as the repair of the fracture may be at times confusing. After determining that the tumor contains new bone production it must then be considered what is the character of the new bone; is it laid down in any definite way? All the bone producing tumors are either cortical or periosteal in origin and in their growth extend out into the soft tissues. In this region bone production is best studied, as here it has free growth, not being confined or hampered by the bone nor can the new bone growth be confused with the bone itself, as might be done if the bone production in that portion of the tumor lying within the bone were considered. Close inspection of this new bone gives the rather startling information that in malignant growths, bone is laid down in long striae practically perpendicular to the shaft, while in benign growths (with the exception of an osteoma) the growth of new bone is more or less parallel to the shaft.

This second cardinal point helps materially in diagnosis by the process of elimination. But there still remain a number of tumors which must be diagnosed, particularly those of medullary origin, so the third cardinal point is involved.

Cortex. It must be determined whether the cortex is present or absent, and if present whether it is expanded in a spherical or longitudunal manner. When considering the diag-

nosis of tumors based upon the first two cardinal points the tumors have been spoken of as arising from the cortex and periosteum, and the production of bone in them has been mentioned; but it is well to check them up again with this third point. The condition of the cortex is also a very important factor. Experience has shown that benign tumors arising in the medullary canal are slow growing and always take the path of least resistance; that is, while they grow slowly toward the



FIG. 253.—Benign tumor extending up and down the medullary canal with slight expansion of the bone. (Cyst.)

cortex they progress more easily up and down the shaft. The pressure from the growth, however, causes an expansion of the cortex, but the expansion is always spindle-shaped (Figs. 253 and 254), or cylindrical, and the cortex is intact unless the growth is unusually large. On the other hand in malignant conditions, the growth is spherical in nature and extends equally in all directions. On account of the rapid growth of the



FIG. 254.—Benign tumor of the fibula. Note the extension within the medullary canal and expansion of the cortex. (Cyst.)

tumor, the cortex does not expand, but the growth involves and destroys it, so that in this lesion the cortex is absent, Fig. 255. In the early stages of such a tumor the growth has not reached the cortex and it is intact; however, since at this time there will be no symptoms, the lesion will not be seen in this stage. It is only after pressure symptoms begin that the patient



FIG. 255.—Complete destruction of the head. The cortex destroyed. (Round-cell sarcoma.)

presents himself for examination. Then these cortical changes are discovered. There is only one exception to this and that is the giant-cell sarcoma, Fig. 256. As the name implies it belongs to the sarcomatous group, but on account of the character of its cells, metastasis is almost impossible, so that practically a benign condition is dealt with.

The fourth cardinal point, invasion, is still to be considered.

Invasion. This point, frequently the hardest to determine, is the most important of all. If it can be definitely established that the growth is invasive, by that meaning that it infiltrates into bone and soft tissue, everything is in reality determined, because *malignancy depends upon invasion*, Fig. 257.



FIG. 256.—Marked expansion of the cortex from a medullary tumor. Cortex thinned out, but still visible. (Giant-cell sarcoma.)

After determining these four cardinal points it is then well to take up what might be termed the three laws of probabilities. By these laws is meant what is most frequently found when the age and the sex of the patient are taken into consideration with the particular bone involved.

Law as to Age. It is known that certain tumors are most



FIG. 257.—Metastatic carcinoma showing invasion. Primary growth in the urinary bladder.

common at some age periods and are very rare at others. Carcinoma is quite common over forty years, that is in the third age period, while it is extremely rare in children, that is in the first age period, and is seen only occasionally between twenty and forty years, the second age period. Sarcoma, however, may occur at any age, but it is most common in the first and second age periods. A malignant tumor of the first age period would then be by this law a sarcoma rather than a carcinoma.

Law as to Sex. The first law demonstrates that carcinoma is a lesion found most frequently in the third age period; it is also known that of all growths in the female at that period, carcinoma of the breast is by far the most common. So in an elderly female, by this law, any growth in a bone is most apt to be carcinomatous in origin.

Law as to Bone Involvement. It has already been stated that carcinoma is a metastatic growth, while sarcoma may be either metastatic or primary in origin. Since carcinoma enters the bones by means of the nutrient canal it generally metastasizes close to the entrance of this canal, while in sarcoma the favorite site for the growth is at the ends of the bone; so again by this law a medullary growth near the middle of the bone will probably be a carcinoma. Again a growth in an elderly female in the thoracic spine is apt to be carcinomatous, first on account of sex, then on account of age, and finally because of all thoracic spine growths carcinomatous metastases of that region are by far the most frequent.

Before attempting to describe the specific lesions of the more common growths, it is of interest to dwell briefly upon how malignant and benign tumors grow and their relation to normal bone. When a malignant growth starts in the medullary canal it grows by cell division. Starting from a central point it grows equally in all directions; consequently such a growth is spherical in shape. It grows as a mass so rapidly that the surrounding bone is killed by pressure and absorbed; the cortex does not have time to expand, but is destroyed. While it is true

that it does extend down the medullary canal, still as a whole it is spherical in shape. It does not penetrate the Haversian canals and does not pierce the cortex but destroys it. Since it grows as a mass it is limited as a mass to one section of the bone, and does not break out in the same bone at another point leaving normal bone in between. Immediately one sees the fundamental differences between it and an inflammatory lesion such as osteomyelitis. In the latter condition the infection spreads by means of the Haversian canals, and consequently destruction of bone may occur at various places with normal bone between. When the infection reaches the cortex it breaks through in one or two places, leaving normal cortex between. This breaking through the cortex relieves the pressure, and the cortex will not be destroyed *en masse* as in growths. In all malignant tumors except periosteal and osteosarcoma there is no new bone production within the tumor or in the bone adjacent to it. In osteomyelitis when the infection subsides there is new bone production at the edges of the infected area, this being nature's way of limiting the infection. This reaction does not take place with malignant growths. The question naturally arises: Why is bone production not seen under such conditions?

It must be remembered that to produce bone there must be a source of stimulation, and that any foreign element introduced into normal bone will produce such a reaction provided sufficient time elapses for the new bone to form. In osteomyelitis the infection is a foreign element, but in the acute stages new bone is not encountered.

The infection travels too rapidly, and sufficient time does not elapse to allow the new bone to form, because the point of stimulation is just where the infection stops and normal bone begins. In a virulent infection, on account of its rapid spread, its point of contact with normal bone is constantly changing its position, and nature does not have sufficient time to lay down new bone because the infection passes over that point and a new contact point is established. It is like attempting to

build a dam by throwing in earth in a rapidly moving stream. The moment the infection subsides, however, its point of contact with normal bone remains practically stationary, and nature then has sufficient time to lay down her dam of new bone and limit the spread of infection. In malignancy there is the rapid growth similar to the acute infection; but the great difference is that at no stage does the rapid growth of the tumor cease as in an infection, for that reason in no period of the growth of the tumor has nature sufficient time at her disposal to lay down a protective dam of new bone. Just as in an infection, the point of stimulation is the point of contact between the tumor and the normal bone.

In benign growths occurring within the medullary canal a totally different condition arises. While the growth starts at a central point as a spherical mass, it grows but slowly, and when it comes in contact with the walls of the bone it expands very slowly in a lateral direction but extends up and down the medullary canal. Its pressure, however, extended laterally, slowly expands and thins out the cortex but does not destroy it as in malignant growths. With the expansion of the cortex going on very slowly there is probably at the point of contact some new bone laid down, just a sufficient amount to counteract the slow pressure destruction from the tumor. With the growth of the tumor the point of contact is slowly pushed out. The result is an expanded but thinned cortex. Since the growth extends down the canal the expansion will not be spherical but cylindrical in shape. The point of contact at the edge of the tumor in the medullary canal will also show a thin line of bone definitely limiting the growth; but, as in the gradual spreading of the cortex, this thin line of bone will also slowly change its position.

The following are the chief characteristics of the more important bone tumors:

CARCINOMA. This is essentially an old age tumor. It is always metastatic in origin, and since it enters by means of the nutrient canal it is most frequently seen at the middle point



FIG. 258.—Metastatic carcinoma showing invasion. Primary growth in the breast. Note the pathological fracture. Impossible to differentiate the points where the tumor ends and the normal bone begins.



FIG. 259.—Small carcinomatous metastases of the skull from a breast tumor.

of the bone, Fig. 258. Since it is epithelial in origin there is no bone production within the tumor, and on account of its rapid growth there is no new bone reaction external to it. It grows equally in all directions as a solid mass, does not expand the cortex but destroys it completely, and shows distinct signs of invasion in the medullary canal and soft tissues.

In the female, carcinoma of the breast is the most common, and its metastases are seen in the order of their frequency in the ribs, thoracic spine, lumbar spine, ilia, femur, especially the greater trochanter, skull (Fig. 259) and humerus. It is almost an axiom that carcinoma of the breast never metastasizes below the elbow and knee.

Carcinoma of the ovaries and uterus seldom metastasizes to bones, but in our series of cases the writers have had two

metastases of the tibia from carcinoma of the urinary bladder. Whether the tibiae are the favorite sites for such lesions it is impossible to say, as two cases are too few upon which to base conclusions. In the male, *carcinoma of the tongue and lip* are



FIG. 260.—Carcinoma of the mandible, secondary to carcinoma of the tongue.

most common and frequently involve the mandible by direct extension, Fig. 260.

Carcinoma of the prostate is also very common and the bones are very frequently involved, Fig. 261 and 262. The bone changes differ slightly from those in other types of carcinoma, but when once seen will always be recognized, as the changes are quite typical. Since this type of tumor grows very slowly, production of new bone is not found within the growth but just at the edge of the growth in the normal bone, in other words at the point of stimulation. Consequently, these metastases seem to be more or less encapsulated in a calcium wall, as nature attempts to limit the growth. There are also irregular areas of increased density of calcium salts throughout the bone where no gross malignancy can be demonstrated. This may be due to malignant infiltration too small to be recognized upon the plate and yet sufficient to produce bone reaction. When a pelvis is so involved it sometimes appears as if the



FIG. 261.—Metastasis from a prostatic carcinoma.



FIG. 262.—The marked density of the lumbar vertebrae with miliary prostatic metastases. This density is due to growth of new bone around the small metastases. The fourth lumbar vertebra has also been slightly compressed. The individual metastases are too small to be seen.

lesion were that of Paget's disease. The bones most commonly affected are the pelvic bones, sacrum, lower lumbar vertebrae, femur and occasionally the scapula and clavicle. Just as in carcinoma of the breast, when the long bones are involved, pathological fractures are frequent. This growth is essentially a lesion of the third age period.



FIG. 263.—Round-cell sarcoma of the humerus. The condition cannot be differentiated from carcinoma except by the position of the tumor in the bone and the absence of a primary carcinomatous growth. Note the destruction of the cortex with no expansion of bone.

HYPERNEPHROMA. This is also of medullary origin and gives the same *x*-ray appearance as carcinoma. In fact it is impossible to differentiate it from any medullary non-bone-producing growth. It does not metastasize to bone with great frequency, but when it does the bones of the upper part of the body are involved, particularly the humerus. It is a lesion

of the latter part of the second and the first half of the third age period.

ROUND-CELL SARCOMA. Like carcinoma this lesion (Fig. 263) is also very malignant. It is medullary in origin, expanding equally in all directions, contains no bone within the growth, does not expand the cortex but destroys it and is invasive. As far as the growth is concerned it cannot be differentiated from carcinoma upon an *x*-ray plate. It may occur at any age period, but is especially common in the first and second age periods. To make a differential diagnosis between such a lesion and carcinoma, indirect evidence must be relied upon, namely, the laws of probability.



FIG. 264.—Spindle-cell sarcoma of the tibia with marked periosteal reaction.

By the law relating to age, if the tumor is in the first age period and the first half of the second period the lesion will be sarcomatous in origin. In the third age period the law does not help us materially, as both types of growth occur.

By the law relating to sex it is known that carcinoma of the breast is by far the most common, and consequently the growth is more apt to be carcinomatous. While prostatic carcinoma is also quite common, yet its bone lesions are typical and it has an identity of its own.

By the law relating to bone involvement, since carcinoma is metastatic entirely, the lesion is more apt to be near the entrance of the nutrient canal. While sarcoma may occur in any portion of the bone, as the primary elements are through-

out the bone, yet the great majority of the lesions are at the ends. Then too carcinoma is but rarely seen below the elbow and knee, and lesions of bones below these two joints are consequently more apt to be sarcomatous in origin.

SPINDLE-CELL SARCOMA. While spindle-cell sarcoma is malignant it does not seem to be as invasive when seen upon an *x*-ray plate. It destroys equally in all directions, is generally medullary in origin, does not expand the cortex, but destroys it. The growth, however, does not show the same degree of invasion and may be mistaken for osteomyelitis, Fig. 264. It gives the appearance of being slightly limited in its extension through the medullary canal.

PERIOSTEAL AND OSTEOSARCOMA. These growths have probably the same origin but they give such a different picture upon the plate that they will be described separately.

Periosteal sarcoma (Figs. 265, 266 and 267), as its name implies, arises from the periosteum. It is a bone-producing tumor, but the major portion of the bone is laid down in the soft tissues. The shaft and periosteum show but little destruction; occasionally the cortex may look a trifle irregular and worm eaten. It is probably the most characteristic of all tumor growths, and when the peculiar way in which the new bone is laid down is once recognized, a mistake in diagnosis will seldom be made. Small striae of calcium salts are laid down approximately perpendicular to the shaft, but not quite reaching the cortex. In other words the lines of bone are laid down in the tumor and apparently are not connected with the periosteum. They resemble, roughly, the picture of the rays of a sunset. In this tumor the greatest growth is in the soft tissues, and the bone changes are entirely outside of the bone proper. This lesion is seen most frequently in the first age period and the first half of the second age period.

Osteosarcoma (Fig. 268) arises from the cortex, and extends out into the soft tissues as well as involving the bone. It is frequently impossible to determine where it arises, as both cortex and apparently the medullary canal are involved. It is a



FIG. 265.—Periosteal sarcoma. An advanced case where the shaft is involved.



FIG. 266.—Periosteal sarcoma showing the faint bone striae in the soft tissue perpendicular to the shaft.

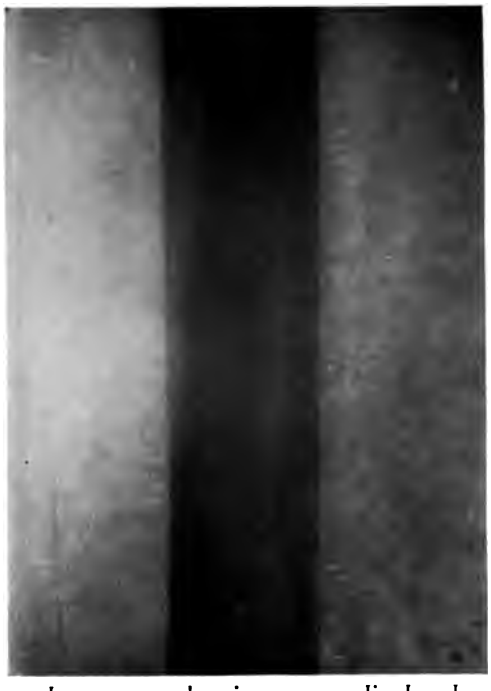


FIG. 267.—Periosteal sarcoma showing perpendicular bone striae in the soft tissues.



FIG. 268.—Osteosarcoma showing the dense bone production. Note the perpendicular arrangement of the bone at the edges of the growth.

bone-producing tumor, and the amount of bone produced depends upon the malignancy of the individual tumor. In the more malignant type there is but little new bone, while in the slow growing type there is marked bone production. Like periosteal sarcoma the growth extends out into the soft tissues, but unlike periosteal sarcoma there is extensive destruction of the shaft. The new bone is laid down in perpendicular striae and is connected with the shaft. When it is laid down massively,



FIG. 269.—Giant-cell sarcoma. Note the expanded but intact cortex and sharp demarcation of the tumor in the medullary canal.

stalagmites of new bone arise from the bone mass like church spires piercing a city's sky line. Of all the malignant growths these are the only two tumors that produce bone within themselves. This lesion is most frequent in the first age period and the first half of the second.

MYELOMA. Myeloma is a malignant tumor which is seen only occasionally. It is slow growing and consequently does not belong to the more malignant class. Its favorite site is the flat

bones, though the long ones may be involved. The cardinal points cannot be applied as a whole in the diagnosis of this tumor. It is a multiple lesion, and the bones are pierced by small focal spots of destruction. It does not expand or destroy the cortex. There is marked rarefaction of the bones; the cortex may be thinned out; pathological fractures are common. It is very difficult to make a diagnosis from the roentgenogram alone; but the clinical history and the presence of Bence-

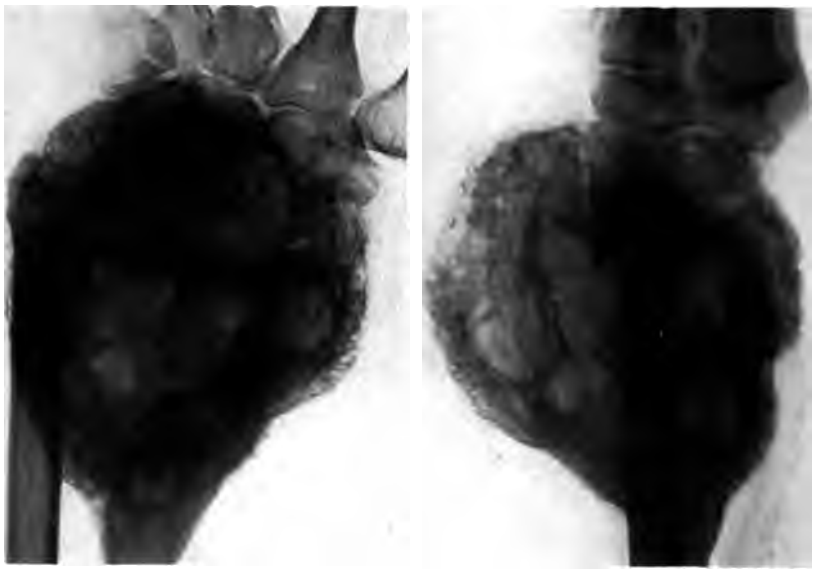


FIG. 270. FIG. 271.

FIG. 270.—A giant-cell sarcoma which underwent spontaneous healing. The growth became calcified.

FIG. 271.—Lateral view of the same condition.

Jones's bodies in the urine with this atypical *x*-ray picture generally lead to a correct diagnosis.

Those tumors will now be considered which are looked upon as benign.

GIANT-CELL SARCOMA. Giant-cell sarcoma (Fig. 269) has a malignant name but is entirely benign in character. This tumor is composed of very irregular cells which in growing are

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF.

so locked together that it is impossible for one to break off and be swept away in the circulation and lodge elsewhere and grow. Occasionally they may heal spontaneously, Figs. 270 and 271. After all, malignancy depends upon two factors—first, the ease with which a cell may break loose from the parent growth, and second, the presence of roads of transportation, ready to carry this cell away and deposit it in some other portion of the body where it grows. The giant-cell sarcoma



FIG. 272.—Multiple enchondromata. These growths are both medullary and cortical in origin. Note how sharply each growth is delimited.

starts in the medullary canal, hence the roads of transportation are there; but the type of cell is such that it cannot become detached from the parent body. Like a malignant tumor it grows equally in all directions; but unlike a malignant tumor it does not destroy the cortex but on account of its slow growth expands it. There is no new bone formation, but the growth

seems reticulated. It is generally limited in the medullary canal by a thin bony wall and does not show any sign of invasion. Its favorite site of location is at the ends of the bone. The lower end of the femur, the upper end of the tibia the lower end of the radius and mandible are most frequently involved, though any long bone may be attacked. It is seen most frequently in the last half of the second age period and at the beginning of the third.



FIG. 273.—Osteochondroma of the head of the fibula.

ENCHONDROMA OR OSTEOCHONDROMA. Enchondroma (Fig. 272) or osteochondroma (Figs. 273 and 274), as its name implies, is cartilaginous in origin and is generally seen before the epiphyses unite. Its early recognition is probably due to the fact that pathological fractures are quite common and the patient comes in for fracture and the *x*-ray then shows the tumor. In this connection it may be mentioned that a fracture



FIG. 274.—Large osteochondroma of the femur.

through such a growth will readily unite, but since the growth is still present the fracture will occur again. Repeated fractures in the same area should always be viewed with suspicion as being pathological from some growth. The lesion is seen near the ends of the bone close to the epiphysis but not involving it. When it is medullary in origin it expands and thins out the cortex but does not destroy it, and since the growth takes the path of least resistance, along the medullary canal, it is oblong in shape and the cortex is expanded in a cylindrical manner. There is no new bone formation unless there has been a fracture.

The growth is cystic in character and frequently is loculated. These tumors are supposed to arise from misplaced bits of cartilage, that is, during the growth of the bone a bit of cartilage at the epiphysis may be surrounded completely by bone but does not itself undergo transformation. This condition probably takes place in everyone, but for unknown reasons the bit of cartilage will proliferate in some. As has just been mentioned these misplaced bits of cartilage are common in many bones, and the stimulation, whatever it may be, causes all these bits to proliferate; consequently enchondromata are almost invariably multiple in origin. They may also arise from the cortex, and in that case form blisterlike lesions in that area. Their growth is always sharply limited, having a definite border, and they are not invasive. Any of the long bones may be involved, but the bones of the hand are most frequently affected. It may be mentioned in passing that malignancy in the bones of the wrist, hand, ankle and foot are so extremely rare that they need not be considered. All growths in these parts may be looked upon as benign. These tumors are practically limited to the first age period and are seen most frequently between the ages of five and fifteen years.

Cyst. Cyst (Fig. 275) is most commonly seen at the same ages as the enchondroma, and like it occurs at the end of the bone in the region of the epiphyseal line. It is medullary in origin and extends up and down the shaft; the cortex is intact, but is expanded in a cylindrical manner. It is sharply limited,

has a definite contour and may be loculated, but is generally one large cyst. Cysts are supposed to be multiple, but in the writers' series of cases only one cyst was present in each case though all the bones were examined. It is often impossible to differentiate a cyst from a single lesion of an enchondroma.





FIG. 275. FIG. 276.

FIG. 275.—Bone cyst of the humerus. Note the expansion of the bone and the sharp limitation of the growth. This cyst is multilocular.

FIG. 276.—Fracture of the humerus. The *x*-ray examination through a plaster cast showed, however, that this was a pathological fracture through a bone cyst.

The differential point is that cysts are single lesions, while enchondromata are multiple. Fractures in cysts are quite frequent, Fig. 276.

OSTEOMA. Osteoma (Fig. 277) arises from the cortex and is almost a solid bone tumor with small areas of cartilage. It extends out into the soft tissues entirely and does not involve



FIG. 277.—Osteoma showing typical cauliflower growth.



FIG. 278.—Osteoma.

the bone, though attached to the cortex by a more or less wide bony pedicle. The bone is laid down symmetrically, radiating from the point of attachment. The growth is lobulated and has a cauliflowerlike appearance. The ends of the lobulation are clean cut and sharp and no invasive changes are present. The growths are generally multiple at the point of origin and arise perpendicularly from the shaft. They are usually seen in



FIG. 279. FIG. 280.

FIG. 279.— Table-top type of exostosis.

FIG. 280.—The pencil type of exostosis. These always point away from the nearest epiphysis.

the first age period, and while they may arise from any bone the favorite sites are the upper end of the humerus, lower end of the femur, and the upper end of the tibia. They are not painful, and the patient comes for examination because the mass of bone mechanically interferes with flexion or on account of cosmetic reasons.

Exostoses are overgrowths of bone arising from the cortex, having the same structure as the bone, limited by sharp, straight borders, and generally enping in a more or less blunt point. They are of two kinds, the table-top type (Fig. 279) similar to a broad-angled triangle, the base being attached to the cortex; and the long-pencil type, arising from the cortex at an angle and pointing away from the nearest epiphysis, Figs. 280 and 281. Occasionally the end is capped by a small osteoma. They are benign in character and occur most frequently in the first and second age periods.



FIG. 281.—Exostosis terminating in a small osteochondroma.



FIG. 282.—Fibroma of the phalanx.

FIBROMA. Fibroma (Fig. 282) is a rather rare tumor. It is cystic in character, has a definite border, is oval in shape extending up and down the medullary canal, thins the cortex, contains no new bone and is not invasive. From an *x*-ray standpoint it cannot be differentiated from a cyst or enchondroma.

It occurs as a single tumor and is not multiple as enchondromata. In the only case observed the tumor was in the phalanx of a patient in the first half of the second age period.

MYXOMA. Myxoma is also an unusual tumor. The *x*-ray appearance is similar to that of a cyst. It sometimes



FIG. 283.—Hemangioma. Note the circular calcified bodies in the soft tissues. Bones are not involved.

undergoes malignant degeneration. This tumor occurs in the first age period.

HEMANGIOMATA (Figs. 283 and 284) are fluctuating tumors, cystic in character which involve most frequently the hands, feet and forearms. They are not primarily bone

tumors, but since there is deposition of calcium salts in them, it was thought well to describe them. The *x*-ray examination shows them as large soft tissue swellings. Lying within the tumor are a series of round calcified bodies having concentric rings within them. This appearance is characteristic, and



FIG. 284.—Hemangioma of the hand.

when once observed cannot be mistaken. These tumors are benign in character.

OSSIFYING HEMATOMA. This growth does not belong to the tumor group, as it is inflammatory in origin; but on account of the frequency with which it is clinically diagnosed as sarcoma, it seems well to consider it under bone tumors. We have already discussed this lesion in connection with scurvy. Since



FIG. 285.—Ossifying hematoma of the femur.



FIG. 286.—Myositis ossificans.

in children the periosteum is but loosely attached to the shaft of the bone, hemorrhage beneath it easily separates it from the bone and the hemorrhage will surround the entire bone, from epiphysis to epiphysis. In adults the periosteum is much more firmly attached, consequently, the lesion will be more or less localized. This condition is the result of trauma to the periosteum, generally following a violent blow. In the writers' seventeen cases in adults sixteen were from injuries received in football, and the seventeenth from the kick of a mule. They all occurred on the anterior surface of the femur. The patients were all males in the latter part of the first age period and first



FIG. 287.—Osteosarcoma, showing destruction of the bone which is one of the differential points in distinguishing it from an ossifying hematoma.

half of the second age period. In nearly half of the cases a clinical diagnosis was made of sarcoma.

Apparently what happens is as follows: The violent trauma evidently ruptures a blood vessel in the periosteum; the hemorrhage gradually raises the periosteum until the resulting pressure becomes greater than the blood pressure, and the hemorrhage ceases. A roentgen examination made shortly after the injury has been sustained reveals nothing, since the periosteum is not demonstrable in its normal condition and the cortex of the bone is never involved. At the end of about three weeks calcium salts will be laid down in the periosteum and the hemorrhage beneath it will undergo organization with deposition of calcium salts, Fig. 285. The lesion on account of the periosteum will have a definite border which will be attached to the bone, and the calcium deposits in the hemorrhage will be laid down more or less parallel to the shaft. This condition must be differentiated from myositis ossificans (Fig. 286) and from the two malignant bone tumors, periosteal and osteosarcoma, Fig. 287.

The following points will aid in the diagnosis. In hematoma, since the hemorrhage is limited by the periosteum, there is a definite, sharp calcium border. The shaft of the bone is never involved and the calcium salts in the hemorrhage are more or less parallel to the shaft. In the malignant bone tumors there are no sharp borders, the calcium deposits are laid down perpendicular to the shaft and the shaft is more or less involved. The tumor from hemorrhage follows the shaft and is generally oblong. The malignant tumors are generally circular in character.

Osteitis Fibrosa Cystica. Although osteitis fibrosa cystica is generally classed with tumors, it is also probably of inflammatory origin. It is practically a disease of the first age period. The femur and tibia are the bones most frequently involved, Fig. 288. The striking feature is the deformity produced by the softening of the bones, which frequently results in pathological fractures. When this condition occurs the lesion is extensive, involving the greater portion or even all of the

bone. As in osteomyelitis the length of the bone may be actually increased. When the femur is involved, particularly the upper portion, the neck may be bent almost to a right angle; the trochanter may be so high as to impinge upon the



FIG. 288.—Osteitis fibrosa cystica, showing the lengthening of the tibia. The bowing is due to the softening and lengthening of the bone. The striae of new bone with cysts are characteristic features.

ilium, and abduction be completely limited. The shaft of the femur and tibia are markedly bowed as in rickets.

Besides these deformities the *x*-ray shows other marked changes. The cortex is expanded and thinned, but intact. There

are large cystlike areas in the medullary cavity. The normal bone structure becomes very irregular. Long striae of lessened and increased density occur, enclosing these cystlike formations. Occasionally these cysts may be very small and numerous and the striated bone be the striking feature. The condition is



FIG. 289.—Pressure atrophy of the cranial bones due to internal pressure. This case was one of marked internal hydrocephalus.

always definitely limited and the cysts have sharp borders. It is sometimes mistaken for osteomyelitis or sarcoma.

BRAIN TUMORS. In discussing the bone lesions of tumors it is advisable to draw attention to the changes in the cranial bones in brain tumors.

Unfortunately the *x*-ray evidence regarding brain tumors is unreliable, but there are certain bone changes occurring in advanced cases which must be noted—those occurring from

internal pressure, whether from growth or fluid, Fig. 289. The cranial bones become so much thinned out in certain areas, *i.e.*, over the convolutions, that in extreme cases the plate suggests that the roentgenologist has actually succeeded in *x*-raying the brain itself. In hypophyseal tumors the sella



FIG. 290.—Complete destruction of the sella turcica from pressure of an hypophyseal tumor.

turcica may be expanded and partially destroyed, also due to pressure, Fig. 290. Brain tumors do not metastasize to bones.

In conclusion it must be remembered that these growths and metastases do not give the same appearance in the flat bones, such as the cranial bones or scapula, and so the four cardinal points cannot be applied to pathological conditions in these regions. In fact one cardinal point, invasion, is completely reversed. In metastatic tumors of the cranial bones there will be a sharp punched-out area with an abrupt and

definite border, while in inflammatory lesions the edges of the infected area will be irregular and worm-eaten, suggesting invasion.

Just as in other bone lesions, and particularly in malignant growths, the roentgenologist depends upon bone destruction and production for diagnostic signs and any surgical interference may so completely change them that he will be completely misled. In such cases one should be extremely guarded in making a diagnosis, as the bone destruction may be the result of surgical interference, and bone production the repair of surgical trauma.

Below is a summary of the bone tumors grouped according to the four cardinal points which aid in diagnosis:

1. ORIGIN—Medullary or Cortical
 a. *Medullary*
 Sarcoma
 Carcinoma
 Hypernephroma
 Myeloma
 Fibroma
 Bone cysts
 Enchondroma
 Giant-cell sarcoma
 b. *Cortical*
 Periosteal sarcoma
 Osteosarcoma
 Osteoma
 Enchondroma
 Ossifying hematoma
 Bone cysts (rare)

2. BONE PRODUCTION
 Periosteal sarcoma
 Osteosarcoma
 Osteoma

2. Bone Production
 - Ossifying hematoma
 - Enchondroma and bone cyst (where there has been trauma)

3. Cortex—Expanded or Destroyed
 a. *Destroyed*
 - Sarcoma
 - Carcinoma
 - Osteosarcoma
 - Hypernephroma
 - Myeloma (advanced stage)
 - Periosteal sarcoma

 b. *Expanded, but Intact*
 - Enchondroma
 - Bone cysts
 - Giant-cell sarcoma
 - Fibroma

4. Invasion
 - All malignant tumors show invasion.

CHAPTER XII

THE SPINE

CHAPTER XII

The Spine

WHILE the spine is subject to the same injuries and diseases as other bones, yet for the sake of bringing out more clearly the differential diagnosis between the various lesions it was thought wise to devote a special chapter to it as a whole.

Conformation. The conformation of the spine is somewhat different from other bones, as it acts as a housing for some of the most important structures of the body—the cord and its attendant nerve trunks.

Each individual vertebra arises from three big centers, one for the body and one for each lateral mass, Fig. 291. These unite to form one solid structure. Between each vertebra is a cartilaginous disc giving a clear joint space as with the long bones. On each side of the body are interlocking articulations preventing any lateral slipping, and the entire spine is bound together by a number of very strong ligaments and muscles. It is probably one of the strongest structures in the body, as it not only carries the weight of the body but protects the spinal cord. The vertebral body is composed entirely of cancellous bone, and relatively there is but little periosteum present. The nerve trunks emerge from between the vertebrae.

Divisions. The spine is divided into the cervical, thoracic, lumbar and sacrococcygeal portions. The bodies of the vertebrae increase in size downward until the sacrum is reached. This is mechanically correct, as the lumbar vertebrae have more weight to sustain than the cervical ones.

While the spine functions as a whole, yet the various parts have slightly different action and are slightly modified by other anatomical relations. The same lesions, therefore, in different parts will be somewhat modified.

The *cervical* portion, especially the upper vertebrae, is the most flexible, has the greatest mobility and sustains less weight, consequently any lesion in that portion gives early clinical symptoms by limitation of motion, just as an ordinary joint would; but there is less compression destruction. The *thoracic* vertebrae articulate with the ribs, and these in turn are fixed to the sternum, so in this region there is relatively little movement; and while these vertebrae carry more weight



FIG. 291.—Showing the three centers from which each individual vertebra arises—one for the body and one for each lateral mass.

than those in the cervical region, yet the ribs and sternum act as supporting walls, so that in the thoracic region a lesion is actually subjected to less compression. In this region clinical symptoms are not noticeable so early, and compression changes appear more slowly. The *lumbar* vertebrae have great flexibility, but no supporting walls, and, while they are the largest and strongest, yet a lesion in them gives early clinical signs and shows marked compression changes.

Fifth Lumbar Vertebra. The fifth lumbar vertebra is a very important one and differs from all others as to function, in that it is the end link of a flexible chain and absorbs the shock of the entire spine. It may be likened to a train of cars stopped by a bumper. The first car hits its neighbor and it in turn moves forward and hits the next one, each one absorbing a certain portion of the shock; but the last car is against the bumper, a fixed and immovable object, and so it stands all of the shock which has not been absorbed by the other cars. In the case of the spine the sacrum and pelvis are the immovable bumper, and so the fifth lumbar vertebra has to take all the remaining shock.

CLASSIFICATION OF LESIONS. The lesions of the spine may be divided into age periods just as with other bones and joints; but the diagnostic points are somewhat complicated, because in a measure every lesion has to be considered as more or less affecting both bone and joint and must therefore be considered together and not separately, as a bone lesion or a joint lesion. From this standpoint our lesions are divided into:

1. Those affecting the intervertebral space and articulating surfaces.
2. Those affecting both the intervertebral space and body.
3. Those affecting the body alone.

Before applying these classifications it will be well to describe the separate lesions first and then see how this classification can be applied so as to bring about points of differential diagnosis.

Fractures. These are but rarely seen in children. They occur most frequently between the ages of twenty and forty, that is, the second age period, less frequently in the third age period. They are most common in the male and relatively infrequent in the female. This is due not to structural weakness of the spine but to the more active and hazardous pursuits of the male. The body of the vertebra is most frequently broken the break occurring most often between the fifth cervical vertebra, and the second lumbar, increasing in frequency

from above downward. In the cervical and upper thoracic region one or two vertebrae may be affected, while in the lumbothoracic region generally only one is involved. Fractures of the body are invariably accompanied by more or less subluxation, consequently, dislocation is one of the *x*-ray signs to be looked for when a fracture is suspected.

Fractures of the cervical region (Fig. 292) are more grave than those in the lumbothoracic region, as in the writers'



FIG. 292.—Fracture with subluxation of the second cervical vertebra.

series of cases the mortality was higher; fracture of the laminae are seen most frequently in the cervical region. This is probably due to the fact that the arch formed by them is large and also because from an *x*-ray standpoint they are easier to demonstrate.

Fractures of the Spinous Processes. These fractures are seen in the cervical region. The processes there are longer and more

slender, and for that reason are more easily broken. Fractures of the spinous processes of the thoracic and lumbar vertebrae are either uncommon or the *x*-ray fails to show them.

Various textbooks state that fractures of the transverse processes are infrequent, but in the writers' series they are quite common, especially in the lumbar region. A fall upon the flat of the back may fracture all of them on one side, or a severe twist may fracture one or more processes, Fig. 293.



FIG. 293.—Fracture of the transverse process of the lumbar spine.

Upon two occasions the writers have seen a fracture of the process of the fifth lumbar vertebra following an attempt to make an extra long drive while playing golf. However, it is probably not correct to draw conclusions as to the relative frequency of fractures of various parts of a vertebra since percentages are based upon only such cases as are *x*-rayed, and then too there may be slight cracks which can not be demonstrated. On numerous occasions an *x*-ray examination of the kidneys has resulted in the accidental finding of one or more



FIG. 294.—Scoliosis showing the primary and secondary curves. Note that there are curves but no angulations.

fractured transverse processes. Upon questioning, the patient may remember an injury, but it was apparently so trivial as not to necessitate medical attention.

There is no question but that many of these fractures are overlooked, and that if percentages were based upon the number of fractures that actually occur a totally different condition might be discovered as to the relative frequency of fractures of the body, laminae and transverse and spinous processes.

Besides examining the suspected area for fracture the spine must be viewed as to the curves. There is normally a curve in the lumbar region with concavity outward and one in the thoracic region with convexity outward. There are no lateral curves except in scoliosis (Fig. 294); consequently, while curves are functional, angulations are due either to injuries or to disease. Angulations always mean some pathological process, and a knowledge of whether they point posteriorly or laterally will aid in diagnosis, Fig. 295. When the body of the cervical vertebra is broken the articulating pedicles are broken also, and allow a dislocation, generally backward, producing an angulation. The joint space disappears, and, if the fracture is old, there will be new bone formation.



FIG. 295.—Lateral angulation indicating a pathological condition. This case was a fracture.

Fractures of the Bodies of the Thoracic and Lumbar Vertebrae. On account of the wide intervertebral space in the normal, the changes resulting from these fractures can be more easily demonstrated. The body is more or less compressed

and widened laterally, the intervertebral space is obliterated, and there is a generally lateral angulation, seldom posterior as in tuberculosis. If the fracture is an old one there will be new bone production, again different from tuberculosis (Fig. 296), as in that lesion there is no bone production.



FIG. 296.—A fractured spine with new bone production.

DIAGNOSTIC AIDS. There are, then, five important points upon which to base a diagnosis.

1. Angulation.
2. Lateral deformity.
3. Bone production.

4. Involvement of the intervertebral space and body.
5. Laws of probability as to age.

By angulation is meant a sharp angular deviation; in the cervical region this may be lateral or posterior, but in the thoracic and lumbar regions it is generally lateral.

Lateral deformity is due to the fact that with fracture the body is compressed and spreads laterally.

In an old fracture there is bone production.

The intervertebral space is always obliterated by crushing.

The law as to age establishes the observation that fractures are most frequent in the second age period.

ARTHRITIS. This condition has already been discussed in the chapters on joint lesions, and while the same bone changes occur in the spine as in other joints there are certain modifications which must be noted.

On account of the thickness of the muscles and the rigidity of the ligaments it is not possible to demonstrate periarticular swelling, and there is no fluid present. Atrophy, except in extreme cases, is also hard to demonstrate. There are then only two processes to fall back upon, destruction and bone production, and of these two, bone destruction is hard to demonstrate in many of the cases. In the ordinary joint the destruction of the cartilaginous spaces was indicated by joint narrowing. The vertebrae, however, articulate by small pedicles, and the bodies are separated by the intervertebral discs. While the cartilage of the articulations is destroyed yet the intervertebral space will not be narrowed, as the discs hold them apart. Consequently the condition is not recognized in the early stages but only in the stage where there is bone production. One frequently sees the stiff, rigid, painful, poker back which in the early stages of the lesion is shown by the *x*-ray as a normal spine. Months later bone production will finally appear.

The first *x*-ray change to be noted will be a flattening of the edges of the vertebrae (Fig. 297) where the lateral ligaments are attached. Later at those points exostoses will form, always

pointing towards the adjacent bodies. This process will go on until they actually engage, Fig. 298.

INFECTIOUS ARTHRITIS—*Tuberculosis.* Just as in the other joints infectious arthritis in the spine occurs at any age, and the hypertrophic type in the third age period, the latter type throwing down more bone than the former. These changes are all confined to the intervertebral space, and, as has been



FIG. 297.



FIG. 298.

FIG. 297.—The edges of the vertebrae are becoming sharpened. This is the first change noted in an arthritis.

FIG. 298.—The formation of exostoses taking place along the edges of the vertebrae and lateral ligaments.

explained, there is no narrowing of the joint space as with other joints. Tuberculosis, however, is a notable exception. In the first stage of the disease there is destruction of the articulating surfaces alone. This stage is but seldom recognized. It is only when there begins to be destruction of the body with obliteration of the space that this lesion is recognized. In the advanced cases there are obliteration of the space and

destruction of the body. There is no new bone formation, and in the thoracic and lumbar regions there is posterior angulation. This is due to the fact that the anterior border of the body becomes softened and then is crushed by the body weight, Fig. 299. The posterior portion of the body and the laminae are practically never involved, so that a lateral *x*-ray examination will show that the vertebra has become triangular in shape, the apex being anterior and the base posterior. This gives the characteristic angulation or knuckle, the true tuberculous kyphosis. Therefore in the non-tuberculous cases there is found:

1. No angulation.
2. No lateral deformity.
3. Bone production.
4. No obliteration of joint spaces, no body involvement.
5. The condition occurring at any age according to the type of infection.

While in the tuberculous cases are found:

1. Angulation.
2. Posterior and not lateral deformity as a rule, according to where the lesion is situated.
3. No bone production.
4. Obliteration of joint space and involvement of body.
5. The condition occurring most frequently in children and young adults.



FIG. 299.—Destruction and fusing of two vertebrae with posterior angulation, result of a tuberculous infection.

It must be borne in mind that, as in other joints, when the tuberculous lesion in the spine becomes a mixed infection then we have bone production.

Marie-Strumpell Type. Besides the arthritic types described there are two arthritic lesions which differ markedly and must be looked upon as separate entities. The Marie-Strümpell type of spondylitis deformans occurs generally be-

tween the ages of twenty and forty, that is, the second age period. It involves the entire spine, frequently the hips and shoulders. It does not attack any of the other joints. It starts



FIG. 300.—Marie-Strümpell type of arthritis deformans. Note the complete bony ankylosis of the ligaments.

in the lumbar region and involves progressively the entire spine, occasionally omitting the axis and atlas. The shoulders and hips are the last joints to become affected. It is a slowly

progressing infection the action of which extends over years, Fig. 300. It is probably infectious in origin, though this has not been definitely established. In the early stages the *x*-ray shows



FIG. 301.—Charcot joint of the fourth lumbar vertebra. Note lateral deviation.

only involvement of the lumbar region, calcification of the ligaments being so complete as to result in actual ankylosis, with the vertebrae very atrophic. The entire spine becomes

involved, accompanied by a marked anterior bowing which, in extreme cases, is so pronounced that the upper portion of the spine is at right angles to a line drawn perpendicularly through the sacrum.

When this state is reached practically all the ligaments have become calcified, particularly those situated anteriorly. In fact the entire spine appears as if molten bone had been poured over it. On account of the extreme anterior flexion the anteroposterior *x*-ray examination shows an apparent obliteration of the joint spaces. This, however, is due to the marked bowing which causes the *x*-ray shadows of the vertebrae to overlap. If a lateral examination is made each joint space will be seen to be intact, the bone change having taken place entirely in the ligaments. In the early stage this disease looks like the ordinary infectious arthritis.

ACUTE SPONDYLITIS DEFORMANS. This lesion gives an acute clinical history. From an *x*-ray standpoint it presents the same picture as the Marie-Strümpell type except that progress is more rapid and that the joint infection is not confined to the spine, hips and shoulders. Any joint may be involved. This condition is probably an atypical form of infectious arthritis.

CHARCOT SPINE AND SYRINGOMYELIA. Charcot spine (Fig. 301) and syringomyelia occurring in the third age period show practically the same changes on the *x*-ray plate and cannot be distinguished except with the aid of the clinical history. These lesions are characterized by the obliteration of the intervertebral space, destruction of the body, lateral angulation, deformity, and bony detritus lying around the destroyed vertebrae.

TUMORS. Tumors in the spinal column give the same *x*-ray appearance as when located in other bones. Secondary carcinoma is the most common of all. The lesions of tumors are all confined to the body alone, and as has been so frequently pointed out do not involve the cartilage. Consequently the joint space is intact and there is no angulation or deformity un-

til the lesion is in an advanced state. Sarcomatous and carcinomatous tissue is relatively dense, and so the body weight compresses it slowly. The characteristic picture shows the body of the vertebra much narrowed, sometimes to less than half the width of the other vertebral bodies; the joint space on each side is intact, Fig. 302. Carcinoma is the most common lesion, round and spindle-cell sarcoma next, then osteosarcoma



FIG. 302.—Carcinoma of the lumbar vertebra, showing the joint spaces intact and the vertebra not compressed at this stage.

and finally osteoma and giant-cell sarcoma. Cyst and enchondroma have never been observed in our series of cases.

A brief description of these lesions has only been given to refresh the memory, as they have been described in more detail under their respective heads.

APPLICATION OF CLASSIFICATION AIDS. An attempt will now be made to show how diagnosis may be very materially

helped by the use of the classification given in the beginning of this chapter.

These lesions may be divided into three groups:

1. *Those affecting the intervertebral space, but not obliterating it.* In these lesions the body of the vertebra is intact, and all the changes take place in and around the joint space. In this class will fall the various types of arthritis and the first stage of tuberculosis.

2. *Those affecting both the intervertebral space and body.* Here are grouped what might be termed transitional lesions, as they are passing over from the pure intervertebral space lesion to the pure body lesion. These lesions are relatively few in number and are noted in the destructive stage of tuberculosis, fracture dislocation, and the neuropathic conditions such as Charcot spine and syringomyelia. Osteomyelitis should be placed in this group, but it affects the vertebra so infrequently that it can be disregarded.

3. *Those affecting the body alone.* This group is composed entirely of tumors.

The characteristic changes of the first group are bone production at the edges of the vertebrae and in the ligaments, no narrowing of the joint space, no angulation or deformity, and occurring at different ages according to the type of infection. There is no change in the body of the vertebrae or obliteration of the joint space. There may be mechanical ankylosis from the interlocking of opposing exostoses, as in hypertrophic arthritis, or one or two bodies may actually unite by means of deposits in the lateral ligaments, or there may be complete ankylosis as in acute spondylitis deformans or the Marie-Strümpell type.

The first stage of tuberculosis falls in this class since only the cartilage is involved at this period. The important point to bear in mind is that this group consists solely of joint lesions.

The second group differs from the first by obliteration of the joint space and involvement of the body. The four principal

lesions are the destructive stage of tuberculosis, Charcot spine, syringomyelia, and fracture dislocation.

Tuberculosis occurs most frequently in the first and second age periods. If this disease is present there is angulation, anteroposterior deformity, and no new bone production; but fibrous or bony ankylosis may be present. In the neuropathic lesions, Charcot spine and syringomyelia, angulation and lateral deformity are present; new bone formation is seldom seen, but bony detritus is generally present. These lesions are seldom seen before the third age period.

In fracture dislocation, angulation, lateral deformity, new bone production and bony ankylosis are present. It is most common in the second age period.

The changes in fracture dislocation apply only to the thoracic and lumbar regions, as in the cervical region the displacement is generally posterior instead of lateral.

The third group is differentiated from the first and second by the fact that the body alone is involved. This group is composed of the tumors. These generally occur in the third age period. The chief points are: no angulation until the late stage is reached, seldom displacement, but narrowing of the body with or without bone production according to the type of lesion.

This classification must not be looked upon as one would a rule in mathematics, as it is only approximate in its exactness. It does help, however, very materially, as each point, if established, limits the number of possibilities. The arthritic group of spinal lesions, just as in other joints lesions, is probably the one that offers the greatest difficulty. The etiological factor is unknown in many of the cases, in others the factors are in dispute, and finally the types seem to overlap clinically. The same perplexities that confront the clinician are equally applicable to the roentgenologist. In studying these joint lesions, the trite phrase that Osler used to force home the difficulties encountered in the treatment of arthritis upon us as students, seems more and more applicable: "Gentlemen,

when a case of chronic rheumatism comes in your front door, climb the back fence, as you will get more credit than if you treated the case."

FUNCTIONAL CONDITIONS. Besides fractures, arthritis and growths, there are other conditions affecting the spine which may be termed functional.

Scoliosis. This is not a disease but a deformity, and may be the result of some abnormality either of the spine or of the structures connected directly or indirectly with it. It sometimes is the result of overdevelopment of certain muscles, but in the vast majority of cases the cause remains undetermined. The process is entirely a functional one, and whenever there is a primary curve there is a secondary one, either above or below, curving in the opposite direction so as to maintain the spinal balance.

Some of the causes that have been demonstrated by *x*-ray examinations are as follows: It has already been mentioned that a vertebra develops from three principle centers, one for the body and one for each lateral mass. There are several secondary centers which appear later, but they are not pertinent in this condition. Any overgrowth or faulty development of any one of the principle centers, but especially those for the lateral masses, will produce an asymmetrical vertebra and thus a scoliosis.

Occasionally in the thoracic region a rudimentary triangular vertebra may be inserted on one side between the adjacent vertebrae, and thus produce a scoliosis. A single cervical rib as well as the sacrolization of one side of the fifth lumbar vertebra has also produced this condition.

Where an arm or leg has been amputated or over or undergrowth of these parts has occurred, a scoliosis will result. In certain trades, such as hod carrying, where one side of the hod carrier's body has been used more than the other, scoliosis will occur. Spinal curves are always functional, while angulations are pathological.

Another spinal lesion about which there is much dis-

cussion is the so-called *sacroiliac subluxation*. This condition is not believed to be a real subluxation, but a ligamentous involvement. It is perfectly true that one frequently sees one side of the sacrum higher than the other, suggesting slipping, accompanied at the same time by marked clinical symptoms. Manipulation under anesthesia and fixation by a plaster cast will result in complete recovery, yet a second *x*-ray examination will fail to show any change at all in the position of the sacrum. The sacroiliac articulation is one of the strongest in the body, and its anatomical structure is such that only the most severe trauma could cause it to slip.

While many cases of indefinite pain and discomfort in this region may be due to ligamentous rather than bony changes yet it must be borne in mind that there is at times a forward slipping of the fifth lumbar vertebra. A lateral view of the fifth lumbar and the top of the sacrum in such cases will show the body of the fifth lumbar projecting, anterior to the top of the sacrum. As an example of this condition, a patient was referred for possible kidney stone on account of pain and albuminuria. When the patient was lying extended upon her back there was such an extreme lordosis that after the kidney examination a lateral examination of the lumbosacral region was made. No stone was found, but there was anterior slipping of the fifth lumbar vertebra.

The patient was placed in a plaster cast, and within three days the albuminuria and pain had disappeared. The cast was worn a year and complete recovery ensued. Of course the subluxation was not reduced; the cast simply prevented further slipping and allowed nature to tighten up her ligaments and adapt herself to the altered state of affairs.

Pressure destruction of the vertebrae is occasionally observed, not due to any disease but to direct pressure from tumors lying adjacent to the spine. This is notably true in aneurysms of the thoracic aorta, particularly those arising from the descending portion. This aneurysm lies well posteriorly. It may come into direct contact with the spine, and by

its continued expansion so press upon one or more vertebrae as to bring about an actual pressure necrosis. The same condition



FIG. 303.— Pressure destruction of the lumbar vertebrae from a tumor lying adjacent to it. When the tumor was removed there was complete regeneration of the vertebra.

has been observed in large tumors of the neck and in tumors of the spinal muscles, Fig. 303.

CHAPTER XIII

ABNORMALITIES

CHAPTER XIII

Abnormalities

THE spine is probably the seat of more abnormalities than any other part of the bony structure, and attention has already been called to the fact that while these abnormalities are not pathological in the sense of disease, yet they may produce symptoms most annoying to the patient. For convenience it is well to consider them according to their locations, namely, cervical, thoracic and lumbosacral.

Extra Ribs. In the cervical region the most common abnormality is the presence of extra ribs, Fig. 304. They usually spring from the seventh cervical, and may be either unilateral or bilateral. The former is the most common. It is present from birth although the symptoms—pain and numbness in the arm—usually do not appear until the second age period. The symptoms usually lead to a diagnosis of neuritis, and if the condition be due to pressure from one of these ribs it will not yield to medical treatment; removal of the rib is the only cure.

Cervical ribs, however, may be present without producing any symptoms at all, and in those cases they should not be disturbed. When the abnormality is unilateral the diagnosis is easy (Fig. 305), since the corresponding rib on the opposite side will be absent. The difficulties arise when cervical ribs are present on both sides. They may be mistaken for the first pair of thoracic ribs. This can be determined only by the examination of the entire thoracic spine to ascertain whether there are twelve or thirteen pairs of ribs present. If there are thirteen it is certain that an extra pair of *cervical* ribs are under consideration. The seventh cervical vertebra is similar in *x*-ray appearance to the first thoracic, hence no conclusions can be drawn as to whether the ribs arise from the seventh

cervical or first thoracic vertebra. It might seem more simple to take the entire cervical portion of the spine and upon examination see if there are six or seven cervical vertebrae present. Unfortunately this would not help, even if only six cervicals were found, because the attachments of the ribs are



FIG. 304.—Rudimentary ribs arising from the seventh cervical vertebra.

variable. They may be attached to the vertebrae one too high or one too low. In other words, the first pair of ribs may be attached to the seventh cervical and stop with the eleventh thoracic, or the first pair may arise from the second thoracic and stop with the first lumbar. In this way there may

be six cervical, twelve thoracic and six lumbar vertebrae, or eight cervical, twelve thoracic and four lumbar vertebrae. While these cervical ribs practically always spring from the seventh cervical, the writers have seen one case where a pair arose from the second cervical; the ends being imbedded in the



FIG. 305.—Cervical rib arising from one side only.

tonsils. The tonsils were removed on account of their pathological condition, and at operation a small bit of bone had to be removed from each tonsil. An *x*-ray examination after operation disclosed this curious anomaly—a pair of cervical ribs.

NON-UNION—RUDIMENTARY VERTEBRAE. Congenital non-union of the laminae that form the spinous process is quite common in the seventh cervical and occasionally present in the sixth, Fig. 306. Its importance lies only in the fact that it is sometimes mistaken for a fracture.

The abnormalities of the thoracic vertebrae are relatively uncommon, the most frequent being the congenital non-union of the laminae of the first two thoracic vertebrae. This abnormality is also of importance for the same reason—that it may be mistaken for fracture. Occasionally one sees a rudimentary vertebra, triangular in shape, inserted on one side. This condition invariably results in scoliosis, Fig. 307. It generally occurs in the upper thoracic region and is important in that the resulting scoliosis will not yield to the ordinary corrective

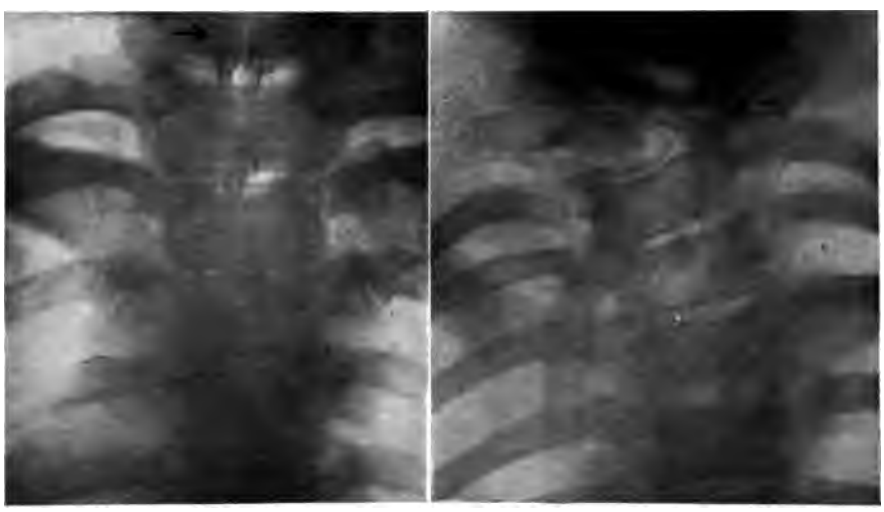


FIG. 306. FIG. 307.

FIG. 306.—Rudimentary spina bifida of the seventh cervical.
FIG. 307.—Rudimentary vertebra in the dorsal region, producing scoliosis.

exercises. Occasionally such a vertebra may have a rudimentary rib attached.

Synostosis of the bodies of two ribs is quite a frequent finding. These are found independent at the spinal attachment; the bodies then become united, but bifurcate again at the costochondral junction, Fig. 308.

ABNORMALITIES IN THE LUMBOSACRAL REGION. In the lumbosacral region nearly all abnormalities are confined to the fifth lumbar and first sacral vertebrae. Congenital non-union

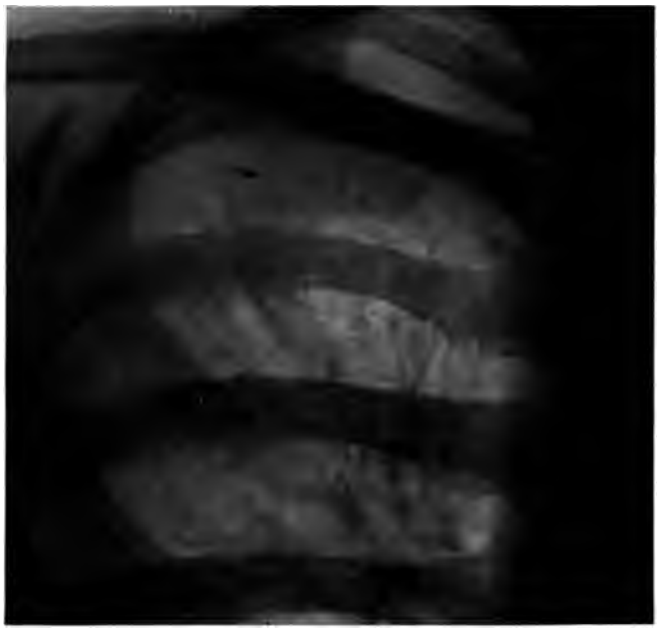


FIG. 308. Synostosis of the bodies of two ribs.

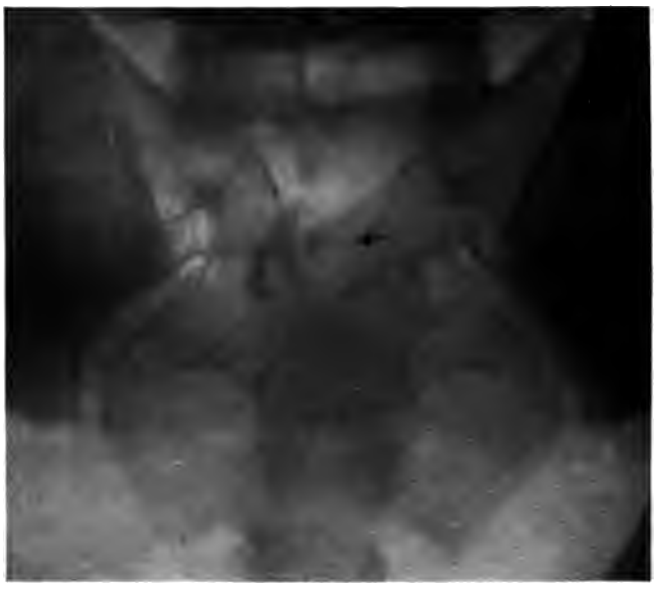


FIG. 309.—Congenital non-union of the laminae of the sacrum.



FIG. 310.—A pair of lumbar ribs.



FIG. 311.—Sacrolization of the fifth lumbar vertebra.

(Fig. 309) is by far the most common abnormality. In a series of 1,000 cases of the lumbosacral region this non-union occurred in over 15 per cent. These were not selected cases with symptoms, but were found in examinations made for kidney and uretral stones, pelvic fractures, etc. In fact every plate upon which this region occurred was included in the series. It



FIG. 312.—An attempt at sacrolization on one side.



FIG. 313.—An aberrant articulation between the clavicle and coracoid. In our series of cases this condition was seen most frequently in the colored race.

should not be mistaken for a fracture. Occasionally a pair of extra ribs will be seen arising from the fifth lumbar vertebra, Fig. 310.

Another abnormality is frequent—the fifth lumbar takes on characteristics of the sacrum. The transverse processes on one or both sides fuse with the sacrum and the fifth lumbar becomes an integral part of the sacrum. This is known as sacrolization of the fifth lumbar vertebrae, Fig. 311. Sometimes the





FIG. 314. FIG. 315.

FIG. 314.—The ulna is not seen due to retarded growth from a faulty epiphysis. Also note the multiple chondroma.

FIG. 315.—An extra digit arising from the little finger.

processes become bulbous and have the shape of the top of the sacrum, but do not unite, Fig. 312. This may be spoken of a pseudo-sacrolization. This abnormality generally gives symptoms. Rudimentary spina bifida of the sacrum is quite common.

CLAVICLE. Occasionally only the sternal portion of the clavicle is present, the acromial end having failed to form. Another interesting abnormality which has been occasionally observed is an articulating facet upon the clavicle forming a joint with the coracoid process of the scapula, Fig. 313. In



FIG. 316.—Fourth and fifth fingers arising from a common metacarpal.



FIG. 317.—Extra digit on the toe, a congenital deformity.



FIG. 318.—Congenital absence of the fibula.

the writers' series of cases this was most frequently found in the colored race. This articulation is normally present in birds.

SCAPULA. The scapula is fairly constant in structure, but occasionally what is known as congenital elevation is seen. In this case the scapula is rotated and elevated. The etiology is unknown, but is probably due to some fetal pressure.

SHOULDER. Most of the deformities of this joint are probably secondary to birth injuries; dislocation and nongrowth of the humerus are also probably secondary to such injuries. The lower epiphysis of the humerus may be affected in a similar manner.

FOREARM. Absence or partial growth of either bone of the forearm Fig. 314 is quite common and occasionally synostosis of the upper third of the forearm is seen.

HAND. Extra digits Fig. 315, fusion of the phalangeal joints Fig. 316, and synostosis of the metacarpals are the most common deformities of the bones, causing club hand.

FEMUR. Congenital absence of this bone is occasionally encountered. Subluxation of the head is sometimes seen following an unrecognized poliomyelitis in infancy. The patella may be either absent or dislocated.

ANKLE AND FOOT. Extra digits Fig. 317, fusion of phalangeal joints, synostosis of the metatarsus and club foot are the most common abnormalities in this region. No attempt is made to enumerate every deformity, only the more common ones that are occasionally met with in x-ray examination.

TIBIA OR FIBULA. Either tibia or fibula or both may be absent or only partially present, Fig. 318.

CHAPTER XIV

DYSTROPHIES

CHAPTER XIV

Dystrophies

OSTEITIS deformans was first described by Paget and is more commonly known by his name. It seems to be a chronic inflammatory process, though the etiological factor is unknown, and it occurs almost entirely in the third age period. A pathological examination shows the bone to be increased in size, sometimes in length, due to a combination of rarefying and proliferative osteitis.

The weight-bearing bones, especially the pelvis, femur and tibia, are most frequently involved; the spine is less frequently affected. The skull when affected is the most typical of all.

The *x*-ray changes are as follows: There are longitudinal striae of porosity and increased density, Figs. 319 and 320. This is due to rarefying and proliferative osteitis. There is proliferation of periosteal bone, causing the bone to become wider. When the rarefying changes are most predominant the bones are weakened and bowing and fractures ensue. In one case under observation for over ten years, where only the tibia was involved, the bone was fractured four times while walking with no additional trauma. Union took place very slowly, about fifteen weeks elapsing each time before there was good union. During the entire process of healing there was but little callus formation. In the leg the overgrowth of the tibia, with fibula unaffected, generally causes a marked bowing.

When the spine is affected the softening of the body of the vertebra with the arches unaffected causes an anterior bowing.

The skull changes are the most characteristic, Fig. 321. The plate will show round, knobbylike masses of bone lying between the inner and outer tables of the bone; sometimes these will be on the outer table, and thus the head will have a knobby irregular feeling when palpated. The best description



FIG. 319.—Osteitis deformans.



FIG. 320.—Osteitis deformans of the tibia. Note the marked bowing of the tibia, due to actual overgrowth of bone. There is a markedly thickened cortex. Striae of bone absorption and bone production are also present.

of this condition is to imagine the curled kinky hair of the negro to have undergone calcification.

The head also increases in size but the enlargement is uniform and does not show any deformity as in acromegaly. This disease is more common in the male than in the female.

ACROMEGALY. This lesion is never seen in the first age period and but rarely in the second. The third age period, or in middle and old age, is the time when it appears or is recog-



FIG. 321.—Changes seen in the cranial bones in osteitis deformans.

nized, Fig. 322. Disease of the hypophysis, especially in derangement of its secretions, seems to be the etiological factor. The roentgenogram shows a general overgrowth of the cranial and, in fact, all the bones. The increased size of the hands and feet is especially noticeable, and there is generally increased tufting of the terminal phalanges giving a clubbed finger effect, Fig. 323. The changes in the head are as follows: There is an overgrowth of the frontal bone, giving what might be



FIG. 322.—Acromegalic skull. Note the overgrowth of bone, prominent frontal sinuses, and the protruding mandible.

termed a beetled brow effect; the frontal sinuses are often markedly enlarged, and the mandible will be lengthened and will protrude so that there will be no occlusion of the front teeth; the sella turcica is frequently much enlarged and very



FIG. 323.—Changes in the bones of the hand in acromegaly. There is increased tufting of the terminal phalanges and increased size of all the bones of the hand.

deep, suggesting an hypophyseal tumor. In gigantism the same picture will frequently be seen.

OSTEOMALACIA. This disease is one of the third age period, and while its etiology is unknown it is supposed to be of an inflammatory nature. It is characterized by an absorption of the calcium salts in an irregular manner; the bones become

softened and often deformed. It is much more common in the female and is frequently associated with pregnancy.

The *x*-ray plate will show the bones to have long striated areas, parallel to the shaft, of diminished density due to absorption of the lime salts. There is no overgrowth of bone. The long weight-bearing bones are bent, and while there are no joint changes yet the altered shape of the bones may change

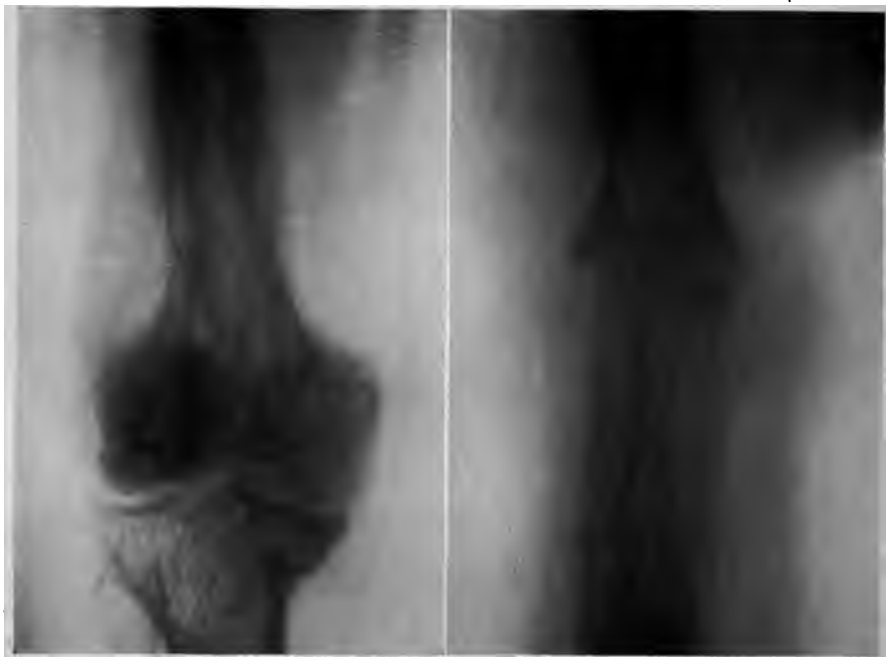


FIG. 324.—Osteomalacia. (*Courtesy of Dr. G. W. Holmes.*)

FIG. 325.—Osteomalacia. (*Courtesy of Dr. G. W. Holmes.*)

the angle of the weight-bearing joint surfaces, which circumstance causes secondary alterations, Figs. 324 and 325.

Fractures are frequent on account of the absorption of lime salts, and they unite slowly and with but little callus. The condition is sometimes mistaken for Paget's disease. These points differentiate: In osteomalacia more bones are involved, the skull remains unaffected, and there is no new bone formation; in Paget's disease the bones of the lower

extremity are most frequently involved; there is bone production, and there are typical changes in the cranial bones.

CHONDRODYSTROPHIES. This disease is of congenital origin and is probably due to some interference or defect in the cartilaginous structure, especially of the epiphyseal cartilages. This results in slow growth of epiphyseal bone, and sometimes complete arrest of growth in certain bones. The periosteum is



FIG. 326.—Achondroplasia.

intact, consequently the changes noted on the *x*-ray plate are limited to the epiphyses. The bones are short, due to poor epiphyseal growth; but since the periosteum is intact it lays down bone in the normal manner, and so the bones are of normal width. The epiphyses also undergo fusion early resulting in abnormally short bones of normal width. At the epiphysis there may be overgrowth of bone, which gives a broadened end to the bone. The ends are often much deformed from aberrant calcification. Practically all the bones are involved, and there is more or less deformity, Figs. 326 and 327.

In cretinism there is a similar change in the bone. In fact, the two conditions are frequently associated.

PULMONARY OSTEOARTHROPATHY. This is an inflammatory disease associated with any chronic infection, especially when the lungs are involved. In the writers' series of cases



FIG. 327.—Achondroplasia with deformity of the epiphysis of the radius from faulty epiphyseal development.

all were associated with pulmonary tuberculosis with the exception of two, one, a primary sarcoma of the pleura, the other a bronchiectasis. The periosteum becomes slightly raised, and there is a deposition of calcium salts in it, but not attached to the bone, Fig. 328.

The *x*-ray plate shows a generalized periostitis particularly of the metacarpals, metatarsals, and the phalanges of the hand and foot. The calcified periosteum is raised, with a clear space between it and the bone, but there is no change in the

bone. The joints frequently show swelling and fluid, but the cartilage is intact. The flesh shadows of the hand show the typical clubbed fingers. In marked cases practically all of the long bones will show this periosteal change. The disease is one of early life and in our series all were in the first age period.



FIG. 328.—Pulmonary osteoarthropathy. Note the periosteal changes.

OSTEOGENESIS IMPERFECTA. This is a childhood infection and is apparently congenital in origin. All the bones may be involved, but especially those of the lower extremity. It is characterized by absorption of the lime salts. The bones on account of the absence of lime salts become so atrophic that it is often difficult to get a satisfactory plate. The joints are never involved. The bones become soft and in part cartilaginous.



FIG. 329.—Osteogenesis imperfecta with multiple fractures. The bones of the lower extremity also suggest achondroplasia.

In such cases there may be distortions at birth, and, on account of the weakness of the bones, multiple fractures are common, Figs. 329, 330 and 331.

A somewhat similar appearance is given by the lesions of rickets and lues, but the differential point is that in the latter disease there are epiphyseal changes, while in the former the epiphyses are intact.

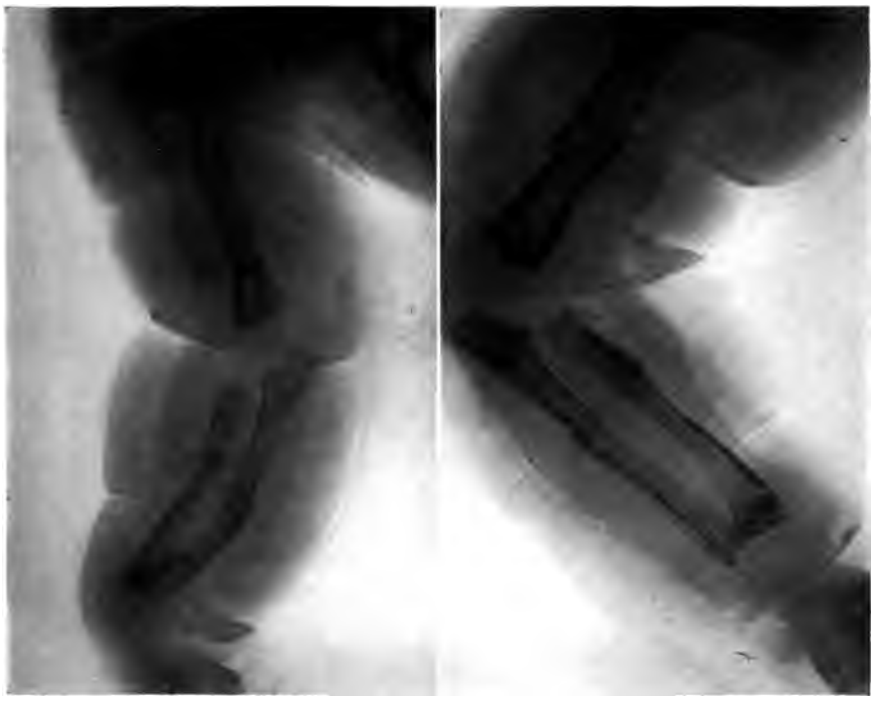


FIG. 330.—Osteogenesis imperfecta, showing multiple fractures.

FIG. 331.—Osteogenesis imperfecta, showing multiple fractures.

When the lesion is seen after the child has walked there will be deformities due to softened bone, and the vertebrae will be very narrow due to flattening from pressure. When the bones are fractured they unite very slowly with but little callus formation.

DYSCHONDROPLASIA. This is also a disease of the cartilaginous structures *in utero*, and as in chondrodystrophia the

epiphyses are involved, so that the bones are shortened and deformed. But there is one feature which does not occur in chondrodystrophia, namely, the formation of multiple exostoses arising from the cortex.

It is often limited to one side of the body. It is more common in the male and frequently successive generations of the same family will show this involvement.

INDEX

INDEX

Abnormalities of the bones, 311
 ankle and foot, 320
 cervical region, 312, 313
 clavicle, 318
 extra ribs, 311
 femur, 320
 fibula, 320
 hand, 320
 lumbosacral region, 314
 rudimentary vertebræ, 313
 scapula, 320
 shoulder, 320
 thoracic region, 314
 tibia, 320
Acetabulum, fracture through the, 95, 96
 mechanical alteration of, in Perthes's disease, 202
 shape of, in congenital dislocation, 135, 137, 138
Achondroplasia, 330, 332
Acromegaly, 325
 cause, 325
 x-ray appearance, 325
Acromial process, centers of ossification of, 35
Acromion, fractures of, 62, 64
Actinomycosis, 176
Acute polyarticular rheumatism, 207
 x-ray appearance of, 208
Acute spondylitis deformans, 302, 304
Age, as factor in, bone development, 27
 diagnosing bone tumors, 242, 251
 joint lesions of children, 183
 production of fracture, 48
 x-ray diagnosis, 4
 laws of probability regarding, 297
 of fracture, estimate of, 53
 relation of, to fractures, 54
Anatomy, normal, necessity for knowledge of, 4
Aneurysms, 8
 of the thoracic aorta in spine involvement, 307, 308
Angulation, in Colles's fracture, 85
 in fractures of the spine, 296, 297
Ankle, abnormalities of, 320
Ankle joint, fractures around, 117
 subluxation of, 139
Ankylosis, as repair process of infectious arthritis, 212
 between patella and femur, gonorrheal origin of, 216
 fibrous, in tuberculosis of the joint, 197
 in fracture of phalanges, 90
 in non-tuberculous hip infections, 137
 in spinal conditions, 304
 in typhoid osteomyelitis of the spine, 175
Arthritic changes due to old age, 222
 in ligaments and bursae, 233
 calcification at bony attachments, 233, 234, 235, 236, 237
Arthritic condition present in elderly individuals, 70
Arthritic lesions of the spine, 305
Arthritis, 206
 indirect evidence of, 206, 207
 infectious, Goldthwaite's classification of, 209
 resemblance of appearance of hemophilia to, 233
 spinal, 223, 297
 See also Chronic arthritis and Joint lesions.
Arthritis deformans, 206, 207
 atrophy in, 207
 indirect evidence of, 207
Astragalus, centers of ossification of, 41
 dislocation of, 156
 fracture of, 120
Atelectatic strip of lung in rickets, 188
Atrophic arthritis, 219
 conditions present, 219
 x-ray appearance, 219
Atrophy, differentiation of local and general, 51
 formed with bone splints, 127, 128

Atrophy, in arthritis deformans, 207
in infectious arthritis, 211
in non-union of fracture, 126
in rickets, 186
of age, 51
of quantity, 51, 138
of quality, 51
presence of as diagnostic aid, in fractures, 49
in gout, 225
usual absence of, in scurvy, 193

Barton's fracture, 86, 87
"Baseball finger," 90
Bence Jones's bodies in urine as diagnostic of myeloma, 266
Benign tumors, brief outline of growth of, 255
direction of growth of, 248
presence of bone production noted in, 6
Blood-vessels, 13, 15, 18
Bone cysts as related to fractures, 54
Bone destruction, diagnosis determined by, 5, 6
causes of, 5
Bone graft in non-union of fracture, 126
Bone infections, *see* Infections, bone.
Bone involvement as factor in diagnosing bone tumors, 253
position of, as diagnostic point in bone tumors, 253
Bone, normal
See Normal bone
Bone production, absence of, in Paget's disease, 328
in tuberculosis of the joint, 197
in malignant growths, 254
as a diagnostic point in fractures of the spine, 296, 297
character of, as diagnostic point, 247
causes of, 5
diagnosis determined by, 5, 6
in arthritis of the spine, 297
in epiphsitis, 200
in mixed tuberculous joint infection, 214
in osteoma, 274
in osteosarcoma, 266
in Paget's disease, 328
in periosteal sarcoma, 263
Bone production in tuberculosis of joint if sinus formation is involved, 198
origin of tumors causing, 247
Bone tumors, 241
Bones, flat, description of, 21
irregular, description of, 21
long, description of, 20
Brain tumors, 282
x-ray appearance of, due to internal pressure, 283
Brody's abscess, 168
Bronchiectasis associated with pulmonary osteoarthropathy, 330
Bursae, arthritic changes in, 233
Bursitis, subdeltoid, 233, 234, 235, 237

Callus, formation of, in fracture, 51, 52
lack of, in osteitis deformans, 323
Cancellous bone, 15
description of, 15
Capitellum, fracture of, 74
Carcinoma, 8, 262
bone destruction in, 5
chief characteristics of, 255
frequency of, in age periods, 253
lack of bone production in, 247
location of bone involvement in, 253
non-union of fracture in, 126
of the spine, 302
origin of, in bone tumors, 242, 246
Caries sicca, 214
x-ray appearance, 215
gouged-out areas, 215
atrophy, 215
Carpal bones, centers of ossification of, 34, 38
dislocation of, 151
Cartilage, 17
description, 17
joint, 17
costal, 18
involvement of, in dyschondroplasia, 333
in chondrodystrophia, 329
in joint injuries, 205
resistance of, to tumors, 241
Centers of ossification, 23, 35
Cervical ribs, extra, neuritis due to pressure of, 311
Cervical vertebra, 290

Charcot joint, 225, 226, 227, 228, 229
neuropathic origin, 225
x-ray appearance, 225–230, 302
periostitis, 231
atrophy, absence of, 231
similarity of gout to, 225
Charcot spine, 302, 304, 305
x-ray appearance, 302
Chauffeur's fracture, 49
Chondrodystrophy, 329
effect of, on epiphyses, 43
cause, 329
x-ray appearance, 329
Chronic arthritis, 209
infectious arthritis, 209
diversified *x*-ray appearance of, 209
first stage, 211
second stage, 211
atrophy, 211
narrowed joint space, 211
third stage, 211
replacement of destroyed area, 211
tuberculosis, 213
x-ray appearance, 213
caries sicca, 214
gonorrheal, 215
luetic, 217
atrophic, 219
conditions present, 219
x-ray appearance, 219
hypertrophic arthritis, 220
x-ray appearance, 221
Clavicle, absence of acromial end of, 318
centers of ossification of, 35
metastasis of, from prostate, 260
spurious facet attached to, 318
Club foot, *x*-ray of, 139
Club hand, due to synostosis of the metacarpals, 320
x-ray of, 139
Clubbed fingers, in pulmonary osteoarthropathy, 331
Coccidoidal granuloma, 177
Cod-liver oil treatment in rickets, metabolic changes from, 195
Colles's fracture, 50, 79, 84, 87
angulation in, 85
dislocation in, 85
impaction in, 85
Colles's fracture, relation of age to, 48
Condyle, fractures through, 59, 79, 109, 112
Congenital abnormalities due to injury of cartilage, 25
Congenital dislocations, 133
Connective tissue origin of sarcoma, 246
Coracoid, fractures of, 62, 148
Coracoid process, centers of ossification of, 35
Coronoid fossa, fracture of, 75
Coronoid process, fracture of, 59, 60, 76
Cortex, 13, 14
description, 14
as point of origin in bone tumors, 243, 245, 247
infection of, known as osteitis, 159
nature of expansion of, in bone tumors, 247
Costal cartilage, 18
Coxa vara, intertrochanteric fracture with, 106, 108
presence of, in Perthes's disease, 202
Cranial bones, changes in, in Paget's disease, 329
Cretinism, association of, with chondrodystrophies, 330
effect of, on epiphyses, 42
Cuboid, centers of ossification of, 41
fracture of, 123, 124
Cuneiform, 38
external and internal, centers of ossification of, 41
fracture of, 123
Cyst, 271
origin, 271
differentiation from enchondroma, 272
example of growth of, 248
Cysts, action of, in expanding cortex, 167
bone, origin of, 246
lack of bone production in, unless accompanied by fracture, 247
relation of, to fracture of mandible, 59

Dakin tubes in compound fracture, 107
Deformity, lateral, as diagnostic point in fractures of the spine, 296, 297
Density, increased, in osteitis deformans, 323

Diagnosis, differential, 6
direct evidences for, bone destruction, 6
bone production, 6
pressure effects, 6
extension of lesion, 6
atrophy, 6, 7
absence of pain, 7
indirect evidence for, age, 8
sex, 8
in bone tumors, 241, 284
in dyschondroplasia, 334
in osteomalacia, 328
in osteomyelitis, 170
in rachitic joint infection, 188
in scurvy, 194
See also under specific injuries and diseases.
Diagnosis, x-ray, elements involved in, 3, 4
See also under specific injuries and diseases.
Diaphyseal side of epiphyseal line involved in lues, 189
Digits, extra, in the toe, 319, 320
Dislocation, from hip injury, 8
in Colles's fracture, 87
of the spine, 304, 305
Dislocations, acquired, 145
shoulder, 145
elbow, 146, 149, 150
ulna and radius, 148, 149
wrist, 151
hand, 151
pelvic bones, 151
hip, 154
patella, 155
knee, 155
fibula, 156
foot, 156
congenital, 133
hip, 133
x-ray differentiation, 134, 136, 137
frequency, 137
shoulder joint, 141
epiphyses, 141
posterior, in Colles's fractures, 85
relation of, to age, 48
Displacement, in subperiosteal fracture, 79
necessity of reporting, 54
of ulna, 76
Dyschondroplasia, 333
Dystrophies, 323
osteitis deformans, 323
acromegaly, 325
osteomalacia, 327
chondrodystrophies, 329
pulmonary osteoarthropathy, 330
osteogenesis imperfecta, 331
dyschondroplasia, 333

Enchondroma, 269
fracture an accompanying feature, 271
nature of growth, 271
differentiation from cyst, 272
origin of, 246
Elbow, fracture of, 76, 77, 146
Endarteritis obliterans involved in Raynaud's disease, 177
Epicondyles, fracture of, 73
Epiphyseal cartilages, defect of, as cause of chondrodystrophies, 329
Epiphyseal centers of the vertebrae, 289, 290
Epiphyseal changes in osteogenesis imperfecta, 333
Epiphyseal flattening in Perthes's disease, 202
Epiphyseal line, changes in, in scurvy, 191
erroneous diagnosis of, 4
expanded in rickets, 184
fractures along, of femur, 99
x-ray appearance of, 24
Epiphyseal separation, erroneous diagnosis of as dislocation, 146
in hip injury, 8
in metacarpals, 88
likelihood of, 48
of pelvis in crushing injuries, 95
of radius as erroneously diagnosed, dislocation, 151
Epiphyses, 13, 23, 33
abnormalities of, 42, 141
value in estimating age, 33
appearance of centers of ossification, 33
variation due to malnutrition, 34, 35
scapula, 35
clavicle, 35
humerus, 36
radius, 38
fracture of, 81

Epiphyses, carpal and metacarpals, 34, 38, 41
phalanges, 38, 41
pubis and ischium, 39
femur, 39
displaced in fracture, 109
patella, 40, 42
tibia, 40, 42
tuberculosis in, 171
fibula, 40
os calcis, 41
astragalus, 41
cuboid, 41
cuneiform, 41
scaphoid, 41
vertebrae, 41, 43
Epiphysitis, acute, 134, 136
in joints of children, 183, 199
x-ray appearance, 199
sharp outlines, 199
focal spots of disease, 199
ankylosis, bony, 200
bone production, presence of, 200
summary of diagnostic points, 200
Etiology in Perthes's disease, unknown character of, 202
Exostoses, 275
in arthritis of the spine, 297
in early stages of gout, 225
in spinal lesions, 304
multiple, in dyschondroplasia, 334
of os calcis, 233, 237
gonorrheal and non-gonorrheal types, 236, 237
presence of, in infectious arthritis, 211

Facial bones, fracture of, 57
Femur, abnormalities of, 320
centers of ossification of, 39
fracture of, 93, 96, 99
neck of, legal case relating to, 53
neck of, prevalent in old age, 48
involvement of, in osteoma, 274
in osteitis deformans, 323
metastasis of, from prostate, 260
non-union in fracture of, 127
site for giant-cell sarcoma, 269
site for ossifying hematoma in adults, 278
Fibroma, 275
Fibroma, similarity in appearance to cyst or enchondroma, 276
Fibula, abnormalities of, 320
benign tumor in, 249
centers of ossification, 41
dislocation of, 156
fracture of, 113
x-ray appearance of Charcot joint in, 230
Fluid formation in acute polyarticular rheumatism, 207
Foot, abnormalities of, 320
dislocation of, 156
Forearm, abnormalities of, 320
fracture of, 75
Fracture, Barton's, 86, 87
Colles's, 84, 85
definition of, 47
erroneous diagnosis of, 3, 313, 314
from hip injury, 8
of neck of femur, legal case relating to, 53
of styloid of the radius, 87
prevalency of, in old age, 48
multiple, in enchondroma, 270
Fractures, kinds of, 47
elements affecting, 47
muscular tension, 47
age and sex, 48
occupation, 48
study of roentgenogram, 49
condition of soft tissues, 49
bone atrophy, 49
formation of callus, 51, 52
fractures from pathological causes, 53
of the upper extremities, classified according to age, 54
skull, 54
facial bones, 57
mandible, 57, 58, 59, 60
clavicle, 59
scapula, 62, 63, 65, 66
humerus, 64, 67, 68, 71
forearm, 74
wrist, 87
hand, 88
ribs, 91
sternum, 94
pelvis, 94

Fractures, of the lower extremities classified according to age, 99
femur, 99
patella, 109
tibia, 113
fibula, 113
bones of the foot, 120
non-union, 127
bone splints, 128
in osteitis deformans, 323
in osteomalacia, 328
multiple, in osteogenesis imperfecta, 333
of the spine, 291
Frontal bone, overgrowth of, in acromegaly, 325
Functional conditions of the spine, 306

Gastrocnemius muscle, displacement due to, 109
Gigantism, resemblance of *x*-ray appearance of, to acromegaly, 327
Glenoid fossa, fractures of, 62
Goldthwaite, classification of, for arthritic conditions, 209
Gonorrheal arthritis, 215
ankylosis between patella and femur as diagnostic of, 216
Gout, 225
x-ray appearance, 225
swelling, 225
atrophy, 225
punched-out areas, 225, 226
exostosis in early stages, 225
Green-stick fracture, 16, 70, 72, 79, 80
of clavicle, 61
of femur, 108
Grafting of bone in fracture, 126
Growth of tumors, brief outline of, 253
Growths, spread of infection in, 6

Hand, abnormalities of the bones of, 320
bones of, involved in enchondroma, 271
fractures of the, 88
Haversian canals, 6, 160, 165, 254
in cortex, 14
Head, fractures of, 54
Hemangiomata, 276
presence of calcified bodies in, 277
Hematomata, 246
in scurvy, 193
Hematomata, ossifying, 277
inflammatory in origin, 277
connection with scurvy in children, 278
association with severe trauma in adults, 278
differentiation, 278, 280
Hemophilia, 233
clinical appearance simulates infectious arthritis, 233
x-ray appearance, 233
gouged-out areas, 232, 233
hazy, 233
organized blood clots, 233
Hemorrhage, 13
in scurvy, 191, 194
subperiosteal, absence of, in rickets, 188
Hereditary character of dystroplasia, 334
Hip, dislocation of, 133, 152, 153, 154
fracture of, diagnosis of, in different age periods, 8
Hip joint involved in Perthes's disease, 201
Histology, knowledge of, as aid to *x*-ray diagnosis, 4, 5
Howland, 195
Humerus, centers of ossification of, 36
fractures of, 64
injuries of, as cause of dislocations, 141
involvement of, in bone cyst, 272
in osteoma, 274
metastasis of, from hypernephroma, 262
Hypernephroma, 260
origin, 260
Hypertrophic arthritis, 8, 220
x-ray appearance, 221
atrophy, presence of, 221
bony exostoses, 222
new bone formation, 222
mechanical ankylosis, 222
in spinal lesions, 298, 304
Hypophyseal tumors, expansion of sella turcica in, 283
Hypophysis, irregularity in secretion of, as cause of acromegaly, 325

Iliac crests roughened by calcification in arthritis, 233
Ilium, fracture of, 93, 95
Impaction, in Colles's fracture, 85, 86, 87
in fracture of femur, 102

Infantilism, effect of, on epiphyses, 42
Infection, presence of bone production noted in, 6
relation of, to fracture of mandible, 59
spread of, through medullary canal, 17
stages of, in joint lesions, 205
Infections, bone, 159
osteomyelitis, 159
channels of infection, 159
x-ray appearance during various periods, 164-167
acute, 167
chronic, 167
diagnostic points, 170
tuberculous, 170
luetic, 172
typhoid, 175
actinomycosis, 176
Raynaud's disease, 176
leprosy, 177
coccidoidal granuloma, 177
mineral poisoning, 180
special infections, 180
virulent, bone destruction in, 5
Infectious arthritis in the spine, 298
tuberculous, 298
non-tuberculous, 299
See also Chronic arthritis and Joint lesions.
Inflammatory nature of osteitis deformans, 323
Intervertebral space, involvement of, in fractures of the spine, 297
Invasion, as diagnostic point in bone tumors, 251, 252
change of aspect of, in brain tumors, 284
lack of, in osteoma, 272
Involucrum, diagnostic value of, in osteomyelitis, 174, 175
Ischium, centers of ossification of, 39
fracture of, 93, 95

Joint cartilage, 17
susceptibility of, to infection, 241
Joint infection of osteomyelitis, 159
Joint, 21
constituents, 21
synovial membrane, 21
invisibility to *x*-ray, 21
indirect evidence of pathology, 21
Joint lesions, 24, 25
in adults, 205
parts involved 205
x-ray appearance, factors governing, 205
stages of infection, 205
arthritis, differentiation of, 206
acute polyarticular rheumatism, 207
infectious, 209
chronic arthritis, 209
summary of differential points, 222
changes due to old age, 222
in the spine, 223
villous, 223
non-arthritic, 224
gout, 225
Charcot joint, 225
syringomyelia, 232
hemophilia, 233
arthritic changes in ligaments, 233
in children, 183
relation of age to, 183
rickets, 183
congenital lues, 188
scurvy, 191
tuberculosis, 195
epiphysitis (non-tuberculous), 199
Perthes's disease or juvenile deforming osteochondritis, 201
Joint mice in hypertrophic arthritis, 222
Joint space, narrowed, in infectious arthritis, 211
in tuberculosis, 196, 214

Knee, dislocations of, 155
Kyphosis, tuberculous, 299

Laminae of the spine, non-union of, 313
Lane plates, use of, in fractures, 129
Lateral deformity as a diagnostic point in fractures of the spine, 296, 297
Legg of Boston, 201
Leprosy, 177
Ligaments, arthritic changes in, 233
imbedded in cartilage, 17
Lues, 172
lace work type of periostitis, 173
as joint infection in childrn, 183
congenital, in relation to joint lesions in children, 188

Lues, congenital, *x*-ray appearance, 188
multiple involvement, 188
changes in the epiphyseal line, 189
presence of punched-out areas, 190
atrophy, usual absence of, 190
periostitis, 190
summary of diagnostic points, 190
effect of, on epiphysis, 42
similarity of, to osteogenesis imperfecta, 333
to osteomyelitis, 171
Luetic arthritis, 217
conditions present, 217
x-ray appearance, 217
Lumbar vertebrae, abnormalities of, 314
Lumbosacral region, abnormalities in, 314
Lymph vessels, 15, 18
Lymphoid origin of osteomyelitis, 159

Malar bone, fracture of, 57
Malignancy of tumors dependent upon invasion, 251
Malignant tumors, bone destruction in, 5
character of growth of, 248, 253
Mandible, fracture of, 57, 58, 59, 60
involvement of, in typhoid osteomyelitis, 175
lengthening of, in acromegaly, 327
Manubrium, fracture of, 94
Marie-Strümpel type of spondylitis, 223, 299, 304
Medullary artery, 15
Medullary canal, 16
as point of origin in bone tumors, 243, 244
as road for osteomyelitic infection, 165
Medullary infection, known as myelitis, 159
Metabolic changes in rickets resulting from cod-liver oil feeding, 195
Metabolism, faulty, as cause of non-union in fracture, 127
Metacarpal, periostitis of, in pulmonary osteoarthropathy, 330
thumb, a vestigial phalanx, 43
tuberculosis in, 172
Metacarpal bones, centers of ossification of, 38, 41
dislocation of, 151
fractures of, 88
Metastasizing to bone, absence of, in brain tumors, 283
Metastatic infection through the medullary canal, 17
Metatarsals, dislocation of, 156
fracture of, 124, 125
periostitis of, in pulmonary osteoarthropathy, 330
Metatarsus, synostosis of the, 320
Mineral poisoning as cause of bone infection, 180
Muscular tension, 47
Myelitis, definition of, 159
Myeloma, 266
diagnostic points, 266, 267
Myositis ossificans, differentiation of, from ossifying hematoma, 280
Myxoma, 276

Nasal bone, fracture of, 57
Necrosis, evidence of, in fracture, 126
Negro race, presence of articulating facet on the clavicle in, 320
Neuritis due to pressure from extra cervical ribs, 311
Neuropathic conditions of the spine, 304
Neuropathic origin of Charcot joint, 225
Non-tuberculous arthritis of the spine, 299
Non-tuberculous joint infections in children, 183
Non-union, congenital, in the lumbosacral region, 316
frequency of, in fracture of the femur, 102
of fractures, 126
causes, 126, 127
bone splints, 127, 128
of laminae of the spine, 313
Normal bone, 13
constituents, 13
periosteum, 13
cortex, 14
medullary canal, 16
nutrient foramen, 16
cartilage, 17
classification, 18
long bones, 20
flat bones, 21
irregular bones, 21
joints, 21

Normal bone, growth, 22
function of epiphyses, 23
centers of ossification, 23
epiphyseal line, 24
joint lesions in children, 24
developmental variations in various age periods, 26, 27
variations due to sex, 27, 28, 29
Nutrient canal, importance of, in metastatic malignancy, 241
Nutrient foramen, 16
description, 16
entrance for infections, 17

Occupation, as factor in shoulder dislocation of males, 145
relation of, to fracture, 49
Olecranon, centers of ossification of, 37, 38
fracture of, 75
associated with dislocation of the elbow, 148
Os calcis, centers of ossification of, 41
fracture of, 123
Os magnum, 38
Osteitis, definition of, 159
Osteitis deformans, as related to fractures, 54
occurrence, 323
x-ray changes, 323
Osteitis fibrosa cystica, 280
probable inflammatory origin, 280
softening of bones and elongation, 280
x-ray appearance, 281
expansion of cortex, 282
cystlike areas, 282
differential diagnosis of, 282
Osteoarthropathy, pulmonary, 330
inflammatory character, 330
x-ray appearance, 330
Osteochondromata, action of, in expanding cortex, 167
Osteogenesis imperfecta, 186, 331
atrophic condition of bones in, 331
Osteoma, 272, 273
bone production as indicative of, 6
origin, 246, 272
x-ray appearance, 274
multiple character of, 274
in the spine, 303
Osteomalacia, 327
Osteomalacia, as related to fractures, 54
inflammatory character of, 327
x-ray appearance of, 328
differential diagnosis of, 328
Osteomyelitis, acute, 127, 167
association of mandible fracture with, 59
as related to fracture, 54
as sequela in fractured phalanges, 90
channels of infection of, 159
chronic, 167
differentiation between, and malignant tumors, 254
erroneous diagnosis of, for osteitis fibrosa cystica, 282
tuberculous, 170
luetic, 172
spread of infection in, 5
x-ray appearance during various periods, 164–167
Osteosarcoma, 263
origin, 246, 263
bone production in, 6, 264
destruction of the shaft, 264
differentiation of, from ossifying hematoma, 279, 280
in the spine, 303
Os trigonum, presence of, in fracture of the ankle, 122
Ovaries and uterus, carcinoma of, 258

Paget, 311
Paget's disease, erroneous diagnosis of, for osteomalacia, 328
Pain, absence of, in Charcot joint, 231
Painful heels, exostoses of os calcis or, 233, 234, 237
gonorrheal and non-gonorrheal types, 236
Park, 195
Patella, abnormalities of, 320
center of ossification of, 40, 42
fracture of, 109, 155
Pathological causes of fractures, 53
Pathology, value of, in *x*-ray diagnosis, 3, 4, 243
Pelvic bones, dislocation of, 151
metastasis of, from prostate, 260
Pelvis, fracture of, 94
involved in osteitis deformans, 323
of female compared with the male, 29, 30

Pelvis, shape of, influenced by walking, 134
in hip dislocation, 135
Periosteal infection, known as periostitis, 159
of osteomyelitis, 159
Periosteal sarcoma, 263
bone production in soft tissues, 263
bone striae perpendicular to shaft, diagnostic point of, 263
origin of, 246
Periosteum, 13
description, 13
function, 13
x-ray appearance, 14
as point of origin in bone tumors, 243
effect of tearing of, on callus formation, 52
Periostitis, definition of, 159
in rickets, 184
mistakes in diagnosis of, 4
of metacarpals, metatarsals and phalanges in pulmonary osteoarthropathy, 330
presence of, in scurvy of the joints, 193
Perthes, 201
Perthes's disease, 183, 200, 201
description of, 201
x-ray appearance of, 202
clear bone detail, 202
epiphyseal flattening, 202
mechanical changes in acetabulum, 202
etiological factor in, 202
Phalangeal enlargement in acromegaly, 325
Phalangeal joints, fusion of, 320
Phalanges, dislocations of, 151
fractures of, 90, 125
involvement of, in Raynaud's disease, 176
of the feet, dislocation of, 156
periostitis of, in pulmonary osteoarthropathy, 330
Phalanx, presence of fibroma in, 276
"Pigeon breast" of rickets, 187
Pisiform, 38
Poliomyelitis, abnormal hip due to, 140
as cause of subluxation of the head of the femur, 320
Porosity in osteitis deformans, 323
Pott's fracture, 119, 120
associated with dislocation, 156
Pressure destruction, as cause of spinal lesions, 307, 308
Pressure effects, and extension of lesion in differential diagnosis, 6
Proliferation of periosteal bones in osteitis deformans, 323
Pronator muscles, prevention of reduction of ulnar fracture because of, 82
Prostate, carcinoma of, 258
Pubis, centers of ossification of, 39
fracture of, 93, 95
Pulmonary osteoarthropathy, 330
Pulmonary tuberculosis, association of, with osteoarthropathy, 330

Quadriceps attachment to patella, calcification of, 233

Radius, centers of ossification of, 37, 38
chauffeur's fracture of, 49
dislocation of, 148
fracture of, 75, 77, 78, 84, 87, 148
Raynaud's disease, 176
Ribs, abnormalities of the, 311
fracture of, 91
Rickets, 8
as joint infection in children, 183
x-ray appearance, 184
atrophy, 186
chest appearance, 186
multiple involvement, 184
periostitis, usual absence of, 184
saucer-shaped expansion of epiphyseal line, 185, 188
summary of diagnostic points, 188
effect of, on epiphyses, 42
metabolic changes caused by cod-liver oil feeding in, 195
similarity of, to osteogenesis imperfecta, 333
Roentgenogram, definition of, 3
See under specific injuries and diseases for x-ray appearance.
Rudimentary vertebrae, 313

"Saber chin" in rickets, 188
Sacral vertebrae, abnormalities of, 314

Sacralization of the fifth lumbar vertebra, 42, 318
Sacroiliac subluxation, 307
non-existence of, 151
Sacrum, metastasis of, from prostate, 260
Sarcoma, 8
absence of bone production in, 247
associated with osteoarthropathy, 330
bone destruction in, 5
confusion of ossifying hematoma with, 280
connective tissue origin of, 246
erroneous diagnosis of, for fracture, 61
for hematoma in scurvy, 193
for osteitis fibrosa cystica, 282
example of destroyed cortex in, 250
frequency of, in age periods, 253
giant-cell, 267, 303
benign character of, 250
nature of growth, 267, 268
origin, 268
hypothetical exclusion of, in bone tumors, 243
non-union of fracture in, 126
round-cell, 262
origin, 262
similarity in *x*-ray appearance to carcinoma, 262
spindle-cell, 263
x-ray appearance, 263
Scaphoid, 38
fracture of, 87, 88, 123
Scapula, abnormalities of, 320
centers of ossification of, 35, 41
fractures of, 62, 63
metastasis of, from prostate, 260
Scoliosis, 42, 306
causes, 306
curves in, 294, 295
due to rudimentary vertebra, 314
Scurvy, 8
as joint infection in children, 183
connection of, with ossifying hematoma, 278
effect of, on epiphyses, 42
joint involvement in, 191
x-ray appearance, 191
multiple lesions, 191
changes in epiphyseal line, 191
Trümmer zone, 192
Scurvy, joint involvement in, atrophy, usual absence of, 193
periostitis, presence of, 193
hemorrhage, 192, 193, 194
summary of diagnostic points in, 194
Sella turcica, enlargement of, in acromegaly, 327
expansion of, in hypophyseal tumors, 283
Semilunar, 38
dislocation of, 151, 155
fracture of, 88
Sequestrum, presence of, in osteomyelitis, 162, 164, 165, 172, 175
Sesamoids, fracture of, 125
Sex, as factor, in diagnosing bone tumors, 242, 253
in bone development, 27
in incidence of fracture, 48, 49
relation of, to acromegaly, 325
to dyschondroplasia, 324
to osteomalacia, 328
Shoulder, abnormalities of, 320
Shoulder joint, dislocation of, 141
Sinus formation in tuberculosis of joint, result of, 198
Sinuses, frontal, enlargement of, in acromegaly, 327
in tuberculous joints producing infection, 214
Skull, fractures of, 54
displaced, 55
linear, 55
of the base, 55
of the vertex, 56
involvement of, in osteitis deformans, 323
lack of involvement of, in osteomalacia, 328
Skull changes in osteitis deformans, 323, 325
Soft tissues, condition of, in cases of fracture, 219
shadows in, diagnostic value of, 119
Spina bifida, 314, 318
Spine, abnormalities of, 42
arthritis in, 223
conformation of, 289
peculiarities of specific divisions, 289
cervical, 290

Spine, peculiarities of specific divisions,
thoracic, 290
lumbar, 290
fifth, 291
classification of lesions, 291
fractures, 291
diagnostic points, 296
arthritis, 297
x-ray changes, 297
infectious arthritis, 298
acute spondylitis deformans, 302
Charcot spine, 302
syringomyelia, 302
tumors, 302
application of classification aids, 303, 304
functional conditions, 306
scoliosis, 307
sacroiliac subluxation, 307
ligamentous changes, 307
pressure destruction, 307, 308
involvement of, in osteitis deformans 323
in typhoid, 175
of the scapula, fractures of, 62
Spinous processes, fractures of, 292
Spondylitis, Marie-Strümpel type of, 223
Spondylitis deformans, 209
x-ray appearance, 302
acute, 223, 302
Sprain, fracture diagnosed as, 117
Sprains and dislocations, relation of, to age, 48
ligamentous, mistaken for subluxations, 152
Sternum, fracture of, 92, 94
Styloid, of the radius, fracture of, 87
of ulna, fracture of, 81
Subcoracoid fracture of shoulder, 145
Subdeltoid bursa, calcification at, in arthritis, 233, 234, 235, 237
Subdeltoid bursitis confused with fracture of tuberosity of humerus, 69
Subglenoid fracture of shoulder, 145
Subluxation, of the head of the femur, 320
in the sacroiliac region, 307
Subperiosteal fracture, 79
absence of callus and displacement in, 52
Superior maxilla, fracture of, 57
Supracondyloid fracture, 72, 73
erroneously diagnosed as dislocation, 146
of humerus, deposit of callus in, 53
Surgical interference, in fracture, 129
necessity for knowledge of, in bone tumors, 284
necessity for recognition of, 180
Symphysis, fracture of, 96
Symphysis pubis, separation of, in females, 8
in males, 8
Synostosis, of rib bodies, 314
of the metacarpals, 320
Synovial membrane, 21
in joint injuries, 205
swelling of, in acute polyarticular rheumatism, 207
thickening of, in villous arthritis, 223
Syringomyelia, 232, 302
neuropathic origin, 232
x-ray appearance resembles Charcot joint, 232, 302
in the spine, 304, 305

Tendo Achillis, calcification at, in arthritis, 233
tension of, as cause of fracture, 123
Thoracic vertebrae, abnormalities of, 314
differential diagnosis concerning, 8
Tibia, abnormalities of, 320
as site for giant-cell sarcoma, 269
centers of ossification of, 40, 42
dislocation of, 155
fracture of, 113, 126
involvement of, in osteitis deformans, 323
in osteoma, 274
x-ray appearance of Charcot joint in, 230
Tongue and lip, carcinoma of, 258
Trapezium, 38
Trapezoid, 38
Trochanter, fracture through, 103
greater, 39
lesser, 40
position of, in hip dislocation, 134
fractures involving the, 107
Trochlea, fracture of, 74
Trümmer zone in scurvy, 191, 195

Tubercle, tibial, injury to, 114
Tuberculosis, a factor in dislocation of tibia, 155
as osteomyelitic infection, 170
adult cases, 170
children's cases, 170
in infectious arthritis, 213
x-ray appearance, 213
haziness, 213
narrowed joint space, 213
atrophy, 213
in joint lesions of children, 183, 195
x-ray appearance, 196
hazy and indistinct, 196
narrowing of joint space, 196
bone involvement, 196
atrophy, presence of, 196
new bone production, absence of, 197
joint, presence of atrophy in, 211
of the shaft of the radius, 169
of the spine, 298, 304, 305
resemblance of appearance of hemophilia to, 233
Tuberculous dactylitis in children, 171
Tuberosity, of humerus, fracture of, 68, 71
tibial, fracture of, 115
Tumors, as causes of pressure destruction in spinal involvement, 308
bone, 241
constituents involved in neoplastic growths, 241
analysis of, 242
origin, 243
bone production, 247
condition of cortex, 247
invasion, 251
law of age, 251
law of sex, 253
law of bone involvement, 253
growth of, 253
carcinoma, 255
cyst, 271
hypernephroma, 260
Tumors, bone, round-cell sarcoma, 262
spindle-cell sarcoma, 263
periosteal sarcoma, 263
osteosarcoma, 263
myeloma, 266
giant-cell sarcoma, 267
enchondroma or osteochondroma, 269
osteoma, 272
fibroma, 275
myxoma, 276
hemangioma, 276
ossifying hematoma, 277
osteitis fibrosa cystica, 280
brain, 282
malignant and benign, as related to fractures, 54
of the spine, 302
classification of, 305
Typhoid infection of the bone, 175

Ulna, backward displacement of, 76
dislocation of, 148
fracture of, 78–83
fracture of styloid of, 81
Unciform, 38

Vertebrae, centers of ossification of, 41
cervical, 290
thoracic, 290
lumbar, 290
fifth, 291
lower, metastasis of, from prostate, 260
rudimentary, 313
Villous arthritis, 223

Wrist, dislocation of, 150, 151
fractures of, 87

X-ray appearance, *see* under specific injuries and diseases.

Zygoma, fracture of, 57

PAUL B. HOEBER
67-69 EAST 59TH STREET
NEW YORK

LANE MEDICAL LIBRARY

To avoid fines, this book should be returned on or before the date last stamped below.

FEB 3 1922
MAR 1 - 1922
MAR 13 1922
AUG 31 1922
OCT 24 27
MAR 24 '23
APR 1 - 1923
APR 1 - 1923
MAY 29 '23
JUL 16 '23

JUN - 7 '23
OCT 5 - 1923
OCT 20 '24
DEC - 4 '25
NOV 9 '26
JAN 18 1927
FEB 18 '27
AUG - 5 '27
SEP - 5 '27
NOV 25 '27

MAY 15 28
NOV 22 28
DEC - 6 28
OCT 21 '29
OCT 21 1931
DEC - 5 1932
May 4
1934

LANE MEDICAL LIBRARY OF
STANFORD UNIVERSITY
300 PASTEUR ROAD
PALO ALTO, CALIFORNIA

M684 Baetjer, F.H. 51168
B14 Injuries and diseases of
1921 the bones and joints.

NAME	DATE DUE

